

Health Effects in the vicinity of Radio/TV towers and mobile phone base stations

**Dr Neil Cherry O.N.Z.M.
Associate Professor of Environmental Health**

6th September 2002

Neil.Cherry@ecan.govt.nz

**Human Sciences Department
P.O. Box 84
Lincoln University
Canterbury, New Zealand**

Health Effects in the vicinity of Radio/TV towers and mobile phone base stations

Dr Neil Cherry O.N.Z.M¹
Associate Professor of Environmental Health
Lincoln University
New Zealand

Abstract:

There is robust scientific evidence that electromagnetic radiation is a Ubiquitous Universal Genotoxic Carcinogen. If this understanding was applied to the data available in 1982, when IARC declared benzene a Human Carcinogen, then the level of data for RF/MW radiation being a human carcinogen was considerably stronger than that for benzene. A large body of laboratory experiments and epidemiological studies now confirm the hypothesis. The evidence is further strengthened through the biophysics understanding of the EMR Spectrum Principle. This shows that as the carrier frequency increases the dielectric constant declines and the induced tissue electric field and induced current increases significantly. This implies and confirms that all of the health effects found in "electrical workers" will be found at much lower mean exposure levels in the vicinity of broadcast towers. Where studies have been carried out, the adverse health effects have been found. When compared with actual radiation patterns they show a causal effect. This confirms that hypothesis and the toxicology of the signals with a safe level of zero exposure. Hence living in the vicinity of broadcast and mobile phone towers produces Cancer, Cardiac, Reproductive and Neurological (CCRN) health effects. It is highly probable that these adverse health effects will be found in the vicinity of cell sites. Because of the small population numbers around single sites, these effects will only be detectable by studying populations around hundreds of cell sites.

Sleep disturbance in the vicinity of the Schwarzenburg Short-wave radio tower in Switzerland was causally related to the RF exposure through dose-response relationships, experimental confirmation and a measured reduction in melatonin in cows and people. A study in France has already shown an exposure-related dose-response in sleep disturbance (and other neurological symptoms) around cell towers, confirming that the effects are the same from cellphone radiation at residential exposure levels. Cellphone radiation also damages DNA. Therefore it is a serious health hazard, even at residential exposure levels, for all CCRN effects.

Introduction

The World Health Organization (WHO) and the international Commission on Non-Ionizing Radiation Protection (ICNIRP) correctly state that exposure assessment is critical to adequately assess health effects from electromagnetic radiation (EMR). This is vital for carrying out and interpreting epidemiological studies of cancer around radar, radio and TV broadcast transmitter sites. Two vital exposure principles are that the study needs to take into account the radial exposure patterns produced by horizontal and vertical antenna patterns and recognize that the far-field exposure experienced by residents, exposed the whole body and not just one particular organ. A second vital study principle involves the population distribution, especially considering what the population density is near the

¹ O.N.Z.M.: Officer of the New Zealand Order of Merit.

towers. For example, if few people live within 1 km of the tower than very few, if any, cancer cases are expected in the near vicinity of the tower. The third assessment principle is appreciation of the evidence that EMR is genotoxic. Evidence of genotoxicity is given by chromosome aberrations (CA), micronuclei formation (MF), DNA strand breakage (DNA), enhanced oncogene activity (OA), enhanced cell neoplastic transformation (NP) and induced toxic (heat) shock protein activity (TSP). The ICNIRP (1998) assessment fails to consider over 20 studies of EMR induced CA or MF, as well as misinterpreting the epidemiological evidence.

These principles are basic common sense. However when the WHO agency IARC (international Cancer Research Agency) fails assesses the evidence of RF/MW being a human universal carcinogen (causing cancer across many body organs) because the whole body is exposed. Failure to recognize this leads to incorrect assessments by ICNIRP (1998) and Elwood (1999).

This study will assess the evidence based on these basic principles. This shows that the adverse health effects of radiofrequency/microwave (RF/MW) radiation are clear and robustly demonstrated, supporting the hypothesis that EMR is a Ubiquitous Universal Genotoxic Carcinogen. This implies that EMR also damages the brain, the heart, the central nervous system and the reproductive system, causing elevated rates of many diseases. Epidemiological evidence confirms that EMR exposure causes elevated incidence of all these types of disease and death rates.

When assessing cancer from exposure to a chemical there is an open question because it is already known that some chemicals do cause cancer. Hence the question is, "does this chemical produce cancer?". When occupational studies find elevated cancer rates in chemically exposed workers then the chemical is declared a human carcinogen with as little as 5 studies, only two of which show significant elevation of the cancer rate. This was in the case for the IARC assessment of Benzene in 1982. This approach is supported because chemicals are often associated with a specific cancer type. For benzene it was originally leukaemia, predominantly myelogenous leukaemia, IARC (1982). Now we know that Benzene is genotoxic and produces a wide range of cancers, Hayes et al. (1996).

Overview of this paper:

This paper will cover the following topics:

- A summary of the IARC assessment of benzene being a specific human carcinogen in 1982.
- A review of the RF/MW evidence of being a human universal genotoxic carcinogen by 1982.
- The evidence of the EMR spectrum principle.
- The principles of carrying out epidemiological studies around radio and TV towers.
- A review of the results of cancer studies in the vicinity of radio and TV towers.
- A review of the evidence trail to cell phone base stations health effects.

IARC's 1982 Benzene Human Carcinogen Assessment:

In 1974 an IARC review concluded that animal carcinogenicity had not been demonstrated and human studies showed that benzene mixtures resulted in damage to the haematopoietic system with suggestion of leukaemia from several case studies and one Japanese case-control study.

In 1982 the IARC re-evaluation reported that workers and the general public were exposed to Benzene from numerous sources including chemicals and the production and use of gasoline.

Chronic human exposure resulted in serious blood changes, benzene crossing the placenta can causing chromosome aberrations (CAs) in bone marrow and peripheral lymphocytes in individuals exposed to high levels of benzene (> 100 ppm), with some findings showing CAs with exposures as low as 10 ppm, but not consistently. A series of epidemiological studies of benzene exposed workers, both cohort and case-control, showed statistically significant associations between leukaemia (predominantly myelogenous) and occupational exposure to benzene and benzene-containing solvents.

Even though: "There was *limited evidence* that benzene is carcinogenic in experimental animals", "There is *sufficient evidence* that benzene is carcinogenic to man."

Hence with the evidence available in 1982 IARC classified benzene as a human carcinogen.

The acceptance is interesting because the epidemiological evidence was relatively small. Some studies found no elevated leukaemia associated with chronic benzene exposure. e.g. Fishbeck et al. (1978), Ott et al. (1978) and Townsend et al. (1978). Note the full author lists are on behalf of Dow Chemical Company:

- Fishbeck, W.A., Townsend, J.C., and Swank M.G. (1978)
- Ott, M.G., Townsend, J.C., Fishbeck, W.A., and Langer R.A. (1978)
- Townsend, J.C., Ott, M.G. and Fishbeck, W.A. (1978)

This could well have been the result of the failure to apply the Healthy Worker Effect.

Studies showing elevated leukaemia were:

Thorpe (1974) found elevated, SMR = 121, n=8.

Askoy et al. (1974) reported an OR = 2.2, n= 34.

Askoy et al. (1980) reported 8 additional cases, raising the significance but failing to give good personal exposure data.

Infante et al. (1977), in a retrospective cohort mortality study, found a significantly elevated leukaemia rate in benzene-exposed white male workers with SMR = 474, n= 7, $p < 0.002$ for one comparison group and SMR = 506, n=7 for another. No dose-response assessment was made.

Rinsky et al. (1981) extended the Infante et al. study with a more complete case follow-up. They reported 7 leukaemia deaths giving a significantly elevated SMR of 560, $p < 0.001$.

This epidemiological evidence was appropriate in 1982 for IARC to conclude that it was sufficient to classify benzene as a human carcinogen. It is based on six published epidemiologic studies showing increased leukaemia, two of these studies from one factory site with significantly increased leukaemia ($p < 0.002$ and $p < 0.001$) from chronic benzene exposures.

Hence there were five studies showing elevated leukaemia and three showing significantly elevated leukaemia and no dose-response relationships, when IARC decided to declare Benzene a Human Carcinogen in 1982.

Since then many other epidemiological studies have reinforced and confirmed this assessment by providing significant and dose-response elevation of leukaemia and other cancers from chronic low exposure to benzene.

Evidence for RF/MW associated cancer available up to 1982:

Genotoxic Studies:

Heller and Teixeira-Pinto (1959) report that a 5 min isothermal pulsed RF exposure of garlic roots produced serious chromosome aberrations that mimicked the effects of c-mitotic chemicals and ionizing radiation. Thus Pulsed RF generators were recommended as a controlled laboratory method to produce chromosome aberrations.

Stodolnik-Babanska (1972) carried out microwave exposures of lymphoblastoid cells in isothermal conditions so that the effects they observed were from microwave exposures no thermal effects. Significant chromosome damage and micronuclei formation was observed and the cells were transformed to cancer cells.

Baranski and Czerski (1976) reviewed the biological effects of RF/MW exposure. In chapter 4 they outline the studies published up until then showing genotoxic effects. They conclude that chromosome aberrations and mitotic abnormalities may be induced under certain conditions and certain cells as a well-established fact since several reports exist from at least, 5 independent laboratories in the West.

Yao (1978) cited 5 previous studies showing that microwaves significantly damage chromosomes, including Heller and Teixeira-Pinto (1959). He then reports that he exposed living Chinese Hamster's eyes to microwaves. This enhanced opacities in the eyes and significantly increased chromosome aberrations. Yao (1982) exposed rat kangaroo RH5 and RH16 cells to 2.45 GHz microwaves, maintaining the temperature at 37°C in the incubator. Chromosome aberrations became evident after multiple passages through the microwave-exposed incubator. The CAs were significantly enhanced in the RH16 cells after 10 or more passages.

Therefore by 1982 it was well established from multiple, independent studies that non-thermal pulsed RF/MW exposure was genotoxic.

Human Studies:

Goldsmith (1997) reported elevated mutagenesis and carcinogenesis among the employees and their dependents that were chronically exposed to very low intensity radar signals the U.S. Embassy in Moscow in the 1950's to the mid-1970's. For most of the time 1953-May 1976, the external signal strength was measured at $5 \mu\text{W}/\text{cm}^2$ for 9 hours/day on the West Facade of the building where the radar was pointed, Lilienfeld et al. (1978). It is stated that the exposure is fairly smooth and consistent over the study period. To get the full strength of the signal a person would have to stand at an open window on the west side of the building at the 6th floor, Pollack (1979). Hence allowing for the internal signal strengths to be between 20 and 100 times lower, the occupants of the embassy were exposed to a long-term average radar signal in the range of 0.02 to $0.1\mu\text{W}/\text{cm}^2$. Blood tests showed significantly elevated chromosome aberrations in more than half of the people sampled. Leukaemia rates were elevated for adults and children.

Most of the staff, with their families, were in Moscow for only one tour of 2 years. Chromosome aberrations found in blood samples are shown in Table 1.

Table 1: Blood samples showed a high proportion of the staff had significantly altered red and white blood cell counts and well above average chromosome aberrations (CA). The CA data is set out in Goldsmith (1997), citing Jacobson (1969) i.e.

Mutagenic Level	Designator	Subjects, No.
5	Extreme	0
4	Severe	6
3.5	Intermediate	5
3	Moderate	7
2.5	Intermediate	5
2	Questionable	5
1	Normal	6

Comparing the 11 level 1 and 2 reference group with the 23 cases above gives:

$$\text{RR} = 2.09, 95\% \text{CI: } 1.22\text{-}3.58, p=0.004$$

Because of the extensive employee concern about the possible health effects of the chronic radar exposure during their service in Moscow, the U.S. State Department contracted Professor Abraham Lilienfeld, Professor of Epidemiology at Johns Hopkins University, to carry out an epidemiological study of staff and dependents. The staff of other US Embassies in Eastern Europe were also surveyed. Health effect rates were compared with the general US population of the similar sex and age range. The sickness and mortality rates for the Moscow Embassy staff and families are summarized in Table 2.

U.S. State Department Embassy employees are careful selected, including physical fitness. This is one basis of the Healthy Worker effect. For these employees, including non-State Dept employees at the Embassy, their overall mortality rate was 47% of the general US population of similar ages. Their cancer mortality rate was 89%. The overall mortality rate suggests a HWE factor of 2. In Table 2 including the "employees" dilutes the health effects significantly.

Table 2: Increased cancer mortality, accidents and suicide in the staff and dependents at the US Embassy in Moscow shown by data in Lilienfeld et al. (1978). This is associated with chronic extremely low intensity (distant and indoor) radar signals. "Adult" refers to adult dependant. # refers to the HWE adjustment. Note (*) p<0.05)

Cancer Site	Observed	Expected	SMR
All Cancer			
Employee	17	19.0	0.89
Employee Adjusted#	17	9.5	1.79
Adult	12	4.5	2.67*
Childhood	4	1.33	3.01*
Total Dependants	16	5.83	2.74*
Total#	33	15.33	2.15*
Leukaemia:			
Employee	2	0.8	2.5
Employee Adjusted#	2	0.4	5.0
Adult	0	0.18	-
Childhood	2	0.5	4.0*
Total Dependants	2	0.68	2.94
Total#	4	1.08	3.70
Breast Cancer:			
Employee	2	0.5	4.00*
Employee Adjusted #	2	0.25	8.00*
Adult	1	0.93	1.08
Total Breast Cancer#	3	1.18	2.54
Adult Hodgkins disease	1	0.07	14.29
Adult Lung Cancer	1	0.56	1.79
Adult Brain Cancer	2	0.15	13.33
Accidents:			
Employees	6	11.6	0.52
Employee adjusted#	6	5.8	1.03
Adults	6	1.39	4.32*
Children	5	6.8	0.74
Total Dependants	11	8.19	1.34
Total#	17	13.99	0.86
Suicide:			
Employees	0	3.9	-
Employees Adjusted#	0	1.95	-
Adults	1	0.56	1.79
Children	1	0.59	1.70
Total Dependants	2	1.15	1.74
Total#	2	3.10	0.65

The employee expected mortality data were adjusted by a factor of 2 for the Healthy Worker Effect (HWE). Table 2 demonstrates how a human population that was chronically exposed low level microwave, with significantly elevated chromosome aberrations, showed significant elevation and elevation of cancers in multiple sites, even though the exposure period was generally a maximum of 2 years. This is consistent with RF/MW radiation being a Universal Genotoxic Carcinogen.

US Radar Repair Workers:

Zaret (1977) reports that 2 brain tumours (astrocytomas) occurred in a group of about 18 workers servicing radar equipment. In another group of 17 workers exposed to highly pulsed EMR for 7 years there were 5 cancers (2 leukaemia, 2 skin cancer, 1 genitourinary tract cancer). Another group of 3 out of 8 men working on another site developed cancer (2 pancreatic cancers).

The SEER (Surveillance, Epidemiology and End Results) cancer data for the US was used to determine the age-specific (30-34 years) cancer for the 1974 period. For the Astrocytoma brain cancer rate the overall brain/CNS cancer rate was used and multiplied by 20%, which is the proportion of all brain cancers identified as Astrocytomas.

Table 3 supports the hypothesis that RF/MW radiation is a Universal Genotoxic Carcinogen that causes cancer across many body organs when the whole body is exposed. While the radar repairing staff in Table 3 were likely to have experienced close to or including thermal exposures, the data in Table 2 certainly does not involve thermal exposures, the peak measured outdoor exposures being over 2000 times below the thermal threshold, and the indoor exposures being much lower.

The following table sets out the results.

Table 3: Cancer rates in groups cited by Zaret (1977) compared with the cancer incidence rates for males aged 30-34 years in 1974, from the SEER database. [n: cases; N group size; P exposure period; Rate: Incidence Rate; Ref.Rate: SEER reference rate.]

Cancer Site	n	N	P Yrs	Rate (/100,000)	Ref Rate	Risk Ratio	95%CI	Fisher Exact p-value
Astrocytoma	2	18	10	1111.1	0.68	1634	385-6939	0.0000009
Skin Cancer	2	17	7	1680.6	0.62	2711	641-11462	0.0000003
Leukaemia	2	17	7	1680.6	3.6	467	70-3126	0.0000257
Pancreas	2	8	7	3571.4	0.348	10263	2287-61962	<0.0000001
Urinary	1	17	7	840.3	1.24	678	92-5007	0.00162
All Cancer	10	43	8	2907.0	62.62	46.4	20.4-105.7	<0.0000001

Table 3 is highly supportive of the hypothesis that RF/MW exposure is a Universal Genotoxic Carcinogen, with significant at a causal level, $p < 0.001$.

Korean War Study:

A major study of US Naval servicemen exposed to radio and radar signals during the Korean War, Robinette, Silverman and Jablon (1980), found significant differences among

occupational groups in all end-points studied, Silverman (1979). They carried out a job exposure matrix survey to assess the microwave exposures of 5% of the about 20,000 servicemen in the "high" exposure group. This resulted in significant exposure related dose-response increase of all mortality, trend $p=0.03$ and for respiratory cancer, trend $p<0.05$. This is classically causal on its own, Hill (1965).

Table 4 and the evidence of chromosome damage adds significant support for the hypothesis of RF/MW radiation being a Universal Genotoxic Carcinogen.

The job exposure matrix survey of the "high" exposure groups, ET, FT and AT, showed, Robinette Table 2, that the ET group was generally low exposed and the FT group was generally high exposed. Hence a dichotomy difference of exposed can be used to see if health effects are elevated in high group compared with the low group.

Table 4: The Mortality Rate of the surveyed high group (Hazard Number: 5000+) compared with the very low exposure group (Hazard Number=0), Robinette et al. (1980). RR is the ratio of the High Group MR to the Low Group MR.

Cause of Death	Risk Ratio	95%CI	p-value
All Mortality	1.28	1.01-1.61	0.038
All Cancer	1.54	1.03-2.29	0.035
Respiratory Cancer	2.68	1.09-6.70	0.022
Leukaemia/Lymphoma	1.50	0.72-3.29	0.26
Other Cancers	1.50	0.68-3.36	0.31
Circulatory Diseases	1.26	0.90-1.72	0.18
Other Diseases	3.60	1.50-8.71	0.0013

Table 5 : Comparison of mortality rates in the high exposure AT group (3273) and lower exposure ET group (13078) from Table 5, Robinette et al. (1980).

Cause of Death	ET	AT	RR	95%CI	χ^2	p-value
All Causes of Death	441	198	1.79	1.52-2.11	46.97	$<10^{-7}$
All Causes excl ⁹ accidents	265	101	1.52	1.21-1.91	13.43	0.00025
All Diseases	199	77	1.55	1.19-2.01	10.89	0.0013
All cancer	65	27	1.66	1.06-2.60	5.93	0.025
Respiratory	16	7	1.75	0.72-4.35	1.56	0.21
Brain/CNS (FT/ET)	5	3	2.38	0.57-9.95	1.50	0.22
Skin	3	2	2.66	0.45-15.9	1.25	0.26
Leukaemia/Lymphoma	18	10	2.22	1.03-4.50	4.32	0.038
Other Cancer	8	4	2.00	0.60-6.63	1.33	0.25
Digestive Disease	11	9	3.27	1.36-7.88	7.81	0.0052
Circulatory Disease	100	31	1.24	0.83-1.85	1.10	0.29

It is important to recognize that the reference or control group is not a "no exposure" group but a lower exposure group. The Healthy Worker Effect is much stronger for uniformed services because of the strong physical requirements for military service. Adjusting for both of these effects suggests an adjustment factor of 2 would be quite conservative.

Grouping the job groups into three exposure ranked groups, Low RM and RD; Middle AE and ET and high FT and AT, allows for a possible dose-response relationship, Figure 1.

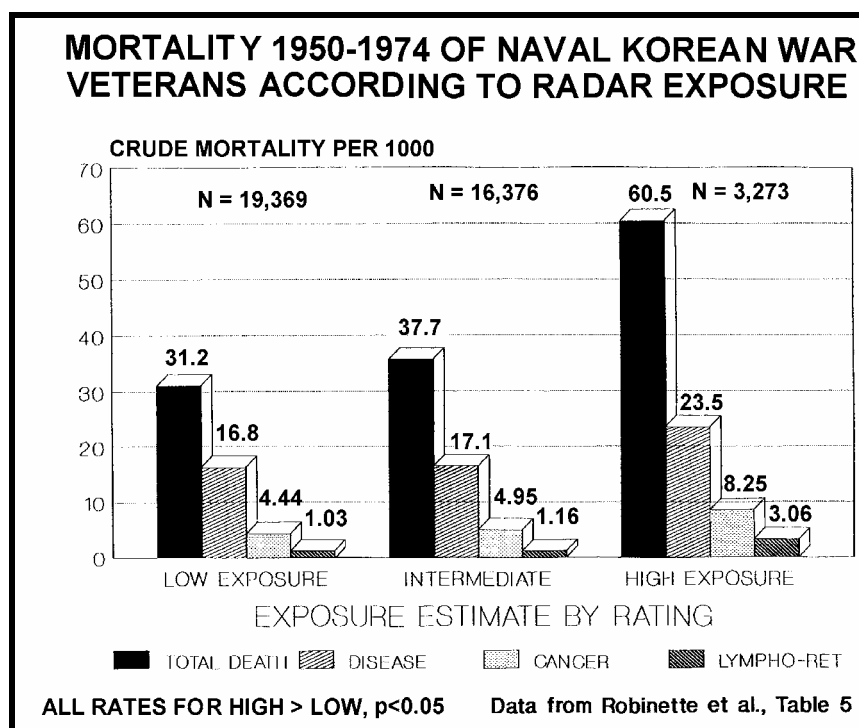


Figure 1: Naval occupations grouped by exposure category, showing dose response increases in mortality for all mortality, all disease, cancer and Lymphatic/Leukaemia. Low exposure (RM+RD), Intermediate exposure (ET+FT), High exposure (AT), prepared by the late Prof John Goldsmith.

Conclusions from Occupational Studies:

Hence up to 1982 there was an established fact that RF/MW radiation is genotoxic (damaging DNA through measurements of chromosome aberrations) with exposure at non-thermal levels. Three studies show that pulsed microwaves from radar chronically exposed people have highly significantly increased cancer in multiple body organs and a dose-response derived from a job-exposure matrix study.

Residential cancer radar-exposure studies:

In 1982 Lester and Moore published a study of radar related cancers in residential populations in Wichita, Kansas, based on a hypothesis that radar could produce cancer. This was based on the evidence of chromosome damage and the Zaret (1977) evidence of cancer rates in radar repairing workers. Because there were airport and air force base radars to the east and west of Wichita they used geographic distributions of total cancer incidence on ridges exposed to both radars, sides of hills exposed to only one radar and valleys sheltered from both radars. Mortality data was obtained from the period 1975-1977.

A significant linear trend ($p=0.034$) was found with incident rates (/100,000 p-yrs) of 470, 429 and 303 respectively from high to low RF/MW exposures, Figure 2. They concluded that their results established a correlation between radar exposure and cancer incidence,

but that more research was necessary for causation. They were unaware of the Moscow Embassy and the Korean War study results that support and confirm their findings.

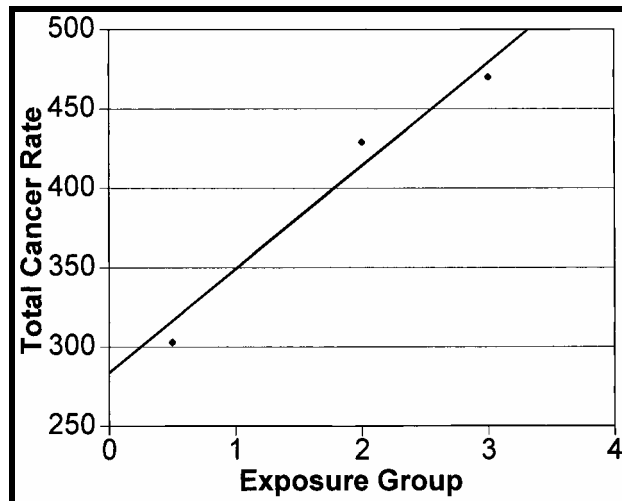


Figure 2: Cancer rates in Wichita, Kansas, for the population not exposed to a radar, exposed to one radar and exposed to two radars, at their residences, Lester and Moore (1982a), Trend $p=0.034$.

They then carried out their own follow-up study to test the hypothesis that cancer mortality is associated, in part, with the possibility of chronic exposure to radar. They studied the cancer rates in 92 counties associated with US Air Force Bases (AFBs) with radars, over the period 1950-1969. They found that counties with AFBs (and radars) had significantly higher cancer rates for males ($p=0.04$) and females ($p=0.02$).

Thus the hypothesis is strongly supported by this study with significant and dose-response increases in All Cancer mortality.

Conclusions from EMR evidence up to 1982:

The evidence that RF/MW radiation is genotoxic is strong, giving a plausible mechanism for a Universal Genotoxic Cancer agent. The human evidence is very significantly higher than for Benzene in 1982, with the level of significance and dose-response relationships at occupational and at residential exposure levels. Hence, if the Universal Genotoxic Carcinogen hypothesis for RF/MW exposure was tested by IARC in 1982, using the same criteria as they did for Benzene, but with the broader view, then they should have declared RF/MW radiation a human carcinogen in 1982.

The evidence is now significantly stronger but the international ICNIRP, IARC and WHO official conclusions get weaker and weaker. This is partly because of the criteria for individual studies and of overall assessments being significantly raised. It is also influenced by the fact that RF/MW and ELF fields are Ubiquitous and we all live in them. This has progressively raised the background cancer rate, making it more and more difficult to identify significant cancer elevation in occupational groups. An exception is cell phone usage, which exposes the user's head to extremely high RF/MW fields, reducing the latency of brain cancer and significantly elevating brain cancer when the position of the aerial and the tumor are compared. Hardell et al. (1999, 2000, 2001, 2002a,b). Hardell et al. (2002b) shows OR = 9.0, 95%CI: 1.14-71, $n=12$, for Astrocytoma from analogue cellphone usage.

This review shows that in 1982 there was much stronger evidence for RF/MW being a Class A, human carcinogen than IARC decided for their benzene carcinogen assessment.

EMR Spectrum Principle:

It is observed that both biological effects and epidemiological effects appear to be the same or very similar from ELF exposure and from RF/MW exposures, including calcium ion efflux, melatonin reduction, DNA strand breakage, chromosome aberrations, leukaemia, brain cancer, breast cancer, miscarriage and neurological effects.

The dielectric constant is approximately the AC equivalent of the DC Resistance. As the dielectric constant decreases the conductivity increases. The dielectric properties of biological tissue depend on the water content because of the interaction of the RF/MW signal with the tissues. Two types of effects control the dielectric constant frequency dependence. One is the oscillation of the free charges or ions and the other the rotation of the molecules at the frequency of the applied electromagnetic signal, Johnson and Guy (1972). This results in a progressive reduction in the dielectric constant with rising frequency of the electromagnetic signal, Figure 3.

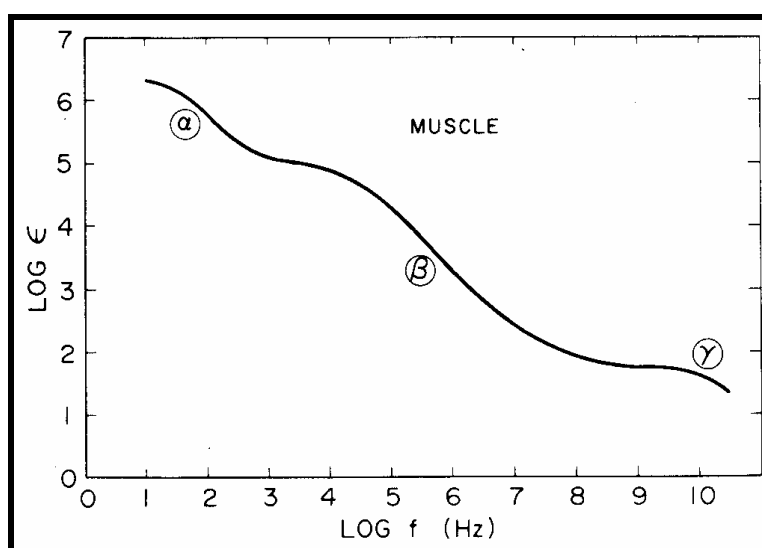


Figure 3: The dielectric constant of muscle as a function of frequency, Schwan and Foster (1980).

The significant drop in dielectric constant with increasing frequency shows a linked process across the spectrum with increasing conductivity and higher induced currents as the frequency rises, Vignati and Giuliani (1997), Figure 4.

Figures 3 and 4 are consistent with data presented by Johnson and Guy (1972). Adey (1988), Figure 5, shows that a 56V/m ELF field induces a tissue gradient of 10^{-7} V/cm, whereas a 56V/m 147MHz signal, modulated by the same spectrum range of ELF fields, induces a tissue gradient of 10^{-1} V/cm, a million times higher. This is a close to the factor given by Figure 4 between 16Hz and 147MHz.

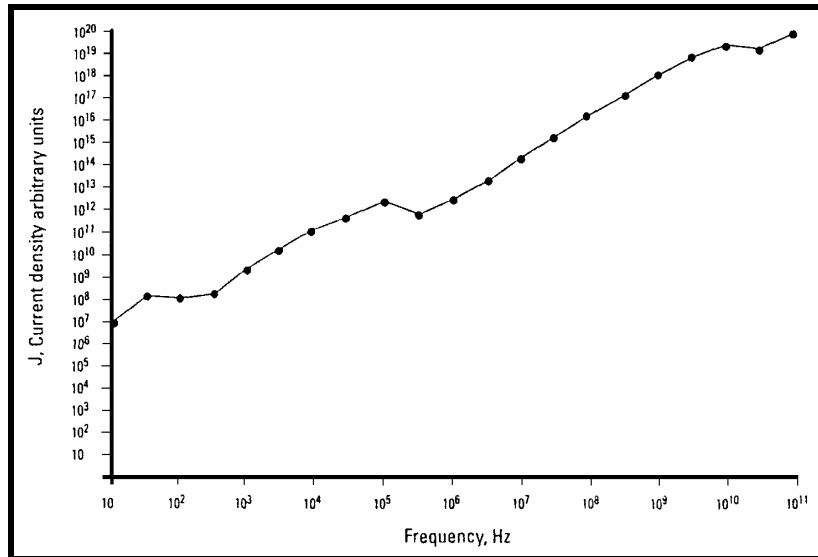


Figure 4: Capacitive induced current density in a toroid of human muscle tissue of unitary radius, exposed to a unitary magnetic field induction, Vignati and Giuliani (1997).

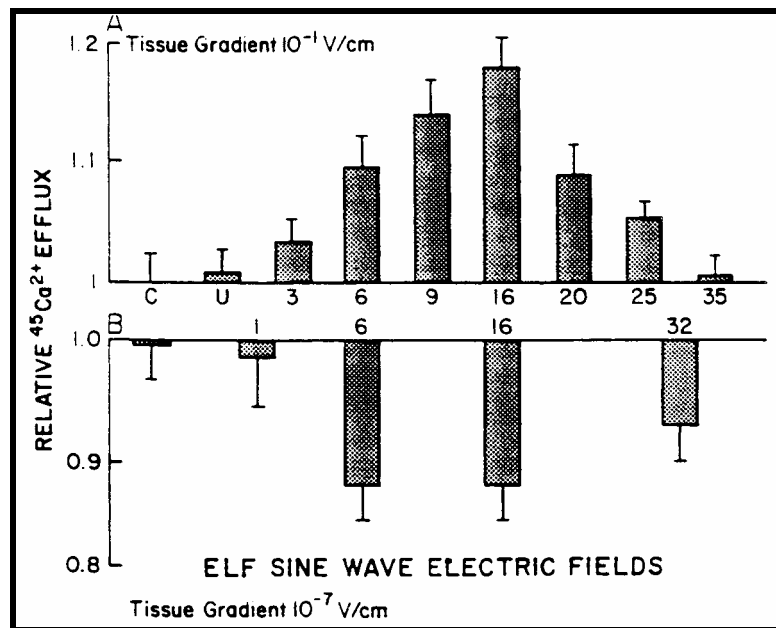


Figure 5: Relative Ca^{2+} efflux (positive and negative) from isolated chick cerebral hemisphere exposed to (A) weak RF field (147 MHz, 0.8 mW/cm², 56 V/m in air), amplitude modulated at low frequencies (abscissa) and (B) ELF electric field (56 V/m in air) over the same ELF modulation frequency, Adey (1988).

The tissue gradients differ by 10^6 between A and B.

Implications of the failure to appreciate the EMR Spectrum Principle:

There is strong evidence of consistent effects across the spectrum and strong support for the biophysical evidence and principles of higher and higher carrier frequencies induced higher and higher currents and induced tissue fields. Therefore effects found from ELF fields are much more likely to occur at much lower mean intensities with exposure to RF/MW fields.

Epidemiological evidence supports the EMR Spectrum Principle:

There is robust and extensive data supporting the EMR Spectrum Principle. While this paper is primarily about RF/MW exposures. Confirmation of adverse effects is given by studies that involve mixed and ELF exposures. Astrocytomas are a subgroup of Gliomas. In a group that was chronically exposed to high ELF fields in electrical utility occupations developed a high rate of Astrocytomas, Theriault et al. (1994).

OR = 28.48 (1.76-461.3)

In a 16-year data set of childhood cancer in the vicinity of the Sutro Tower, Selvin et al. (1992), a powerful radio and TV tower in San Francisco, out to a distance of 1 km the mean exposures are about $0.1\mu\text{W}/\text{cm}^2$. Five brain cancers occurred in a population of about 736 children. This gives:

RR = 31.1 (9.5-101.7), p=0.0000048

Within 500m of the tower there were 2 brain cancers within a population of about 114 children. Their estimated mean personal exposure was about $0.4\mu\text{W}/\text{cm}^2$. This gives:

RR = 80.4 (16.4-394), p=0.00046

Zaret (1977) reports that in a group of 18 workers who were servicing microwave communication equipment there were 2 with Astrocytoma. Allowing for a 10-year exposure and cancer development period, this gives an incidence rate of 1111 per 100,000p-yrs and a relative risk of:

RR = 1634 (385-6939), p<0.0000009

This shows that RF/MW radiation exposure produces very high increased rates of brain cancer. It is even higher for residential exposure levels for childhood brain cancer than compared to high ELF exposures in electrical occupations.

The EMR Spectrum and the data supporting it give robust support for the *a priori* hypothesis is that electromagnetic radiation and ELF fields are a Universal Genotoxic Carcinogen.

It is established in toxicology that a genotoxic substance has no safe threshold level because the damage occurs cell-by-cell. This statement is contained in the UK Royal Commission on Environmental Pollution, Report No. 23, Setting Environmental Standards, UKRCEP (1998).

For genotoxic carcinogenic substances the safe threshold is zero and the approach that should be taken with genotoxic carcinogens is the *de minimis* approach.

Cell phones produce high exposure of the users' head which is clearly not *de minimis* and has already been shown to significantly increase the risk of brain cancer in analogue phone users. The EMR Spectrum Principle, along with the evidence of genotoxicity, shows that the risk of brain cancer, and many other health effects, is elevated in populations living within the vicinity of cellphone base station antennas.

Global Leukaemia dose response for RF/MW exposure:

Epidemiological studies reveal significant elevations of All Cancer and Leukaemia for military occupations exposed to radar and radio, for amateur radio operators and electrical workers exposed to RF signals. As a class of studies military exposures produce high Rate Ratios (RRs), recreational and occupational exposures are intermediate and residential exposures are low. Table 6 summarizes several studies that are ranked in mean exposure order. Military, occupational and residential studies shows a global dose response relationship for increased adult leukaemia and RF/MW exposure with a dose-response threshold close to zero.

Table 6: A summary of epidemiological studies involving adult leukaemia mortality or incidence, ranked by probable RF/MW exposure category.

Study	Reference	Exposure Category	Leukaemia Type	Risk Ratio	95% Confidence Interval
Polish Military (Mortality)	Szmigielski (1996)	High	ALL	5.75	1.22-18.16
			CML	13.90	6.72-22.12
			CLL	3.68	1.45-5.18
			AML	8.62	3.54-13.67
			All Leuk.	6.31	3.12-14.32
Korean War Radar Exposure (Mortality)	Robinette et al. (1980)	High AT/ET	Leuk/Lymp	2.22	1.02-4.81
Radio and TV Repairmen	Milham (1985)	Moderate	Acute Leuk. Leuk.	3.44 1.76	
Amateur Radio (Mortality)	Milham (1988)	Moderate	AML	1.79	1.03-2.85
UK Sutton Coldfield <=2km	Dolk et al. (1997a)	Moderate	Leuk	1.83	1.22-2.74
North Sydney TV/FM towers (Mortality)	Hocking et al.(1996)	Low	All Leuk.	1.17	0.96-1.43
			ALL+CLL	1.39	1.00-1.92
			AML+CML	1.01	0.82-1.24
			Other Leuk	1.57	1.01-2.46
UK TV/FM (Incidence)	Dolk et al. (1997b)	Low	Adult Leuk.	1.03	1.00-1.07

Note: ALL : Acute Lymphatic Leukemia; CLL: Chronic Lymphatic Leukaemia; AML Acute Myeloid Leukaemia; CML: Chronic Myeloid Leukaemia; and All Leuk.: All Adult Leukaemia.

When actual residential exposures are considered in detail, comparing actual radial radiation patterns with cancer patterns, dose responses for residential cancer are also shown by Selvin et al. (1992) (Re-analysis by Cherry), Hocking et al. (1996), Dolk et al. (1997 a,b) and Michelozzi et al. (1998). These show a causal effect of adult and childhood

leukaemia at levels of residential exposure involving exposure levels produced by cell sites out to over 500m.

The Special case of Broadcast Tower Epidemiological Studies:

Two fundamental study methods need to be understood and appreciated in order to carry out or interpret the results of epidemiological studies of health effects in the vicinity of broadcast towers.

- (a) The population pattern in the vicinity of the tower.
- (b) The horizontal radiation exposure pattern, a combination of the horizontal antenna patterns and each antenna vertical radiation pattern.

Three principles are involved in the assessment of health effects around broadcast towers, Cherry (2001):

- (1) Assess the size of the population living in close proximity to the tower because to identify a high cancer rate requires a large population. If few people live near a tower then health effects cannot be detected.
- (2) The horizontal radiation pattern needs to have a high exposure level near the tower if the near tower cancer rate is to be elevated. VHF signals, generally used by AM and FM radio stations, have high exposure levels within 1 km of the tower while UHF signals generally peak outside 1.5 to 2 km from the tower.
- (3) To have a high cancer rate near the tower there needs to be an RF/MW sensitive cancer-type that has a short latency and high rate. These factors must be present in order to raise the number of cases to a detectable level.

The combination of population patterns and radiation patterns lead to two generally radial patterns, Cherry (2001).

Type A Pattern: Low near the tower, rising to an undulating peak between about 1.5km and 6 km. Beyond this the pattern declines with distance.

Type B Pattern: High near the tower, falling in an undulating fashion with distance from the tower.

Broadcast towers provide a unique opportunity for determining whether or not RF/MW exposures are causally related to cancer. This arises from two factors. The first is the large populations that may be exposed and the second is the particular shape of the radial RF patterns. The ground level radial RF radiation patterns are complex undulating functions of the carrier frequency, the height of the tower and the antenna horizontal and vertical radiation patterns. When rates of disease follow these patterns it excludes all other factors, removing all possible confounders.

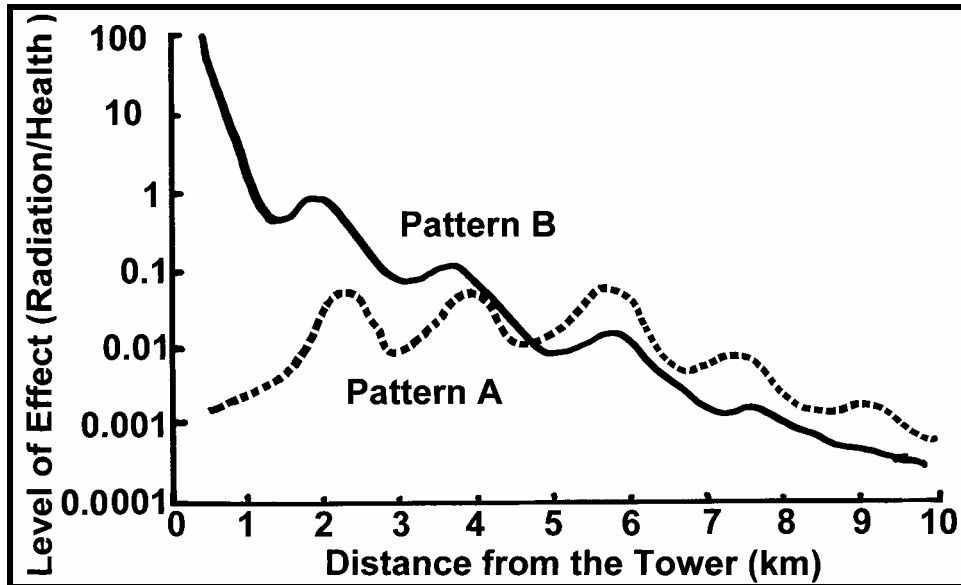


Figure 6: Typical antenna and health effect patterns around broadcast transmission towers. Pattern A (dashed) is typical of UHF antennae and health effects patterns with no VHF and/or low population numbers near the tower.

Failure to understand these study principles and methods leads to the wrong conclusions. For example, Dolk et al. (1997a) found a Pattern B for adult leukaemia in the vicinity of the Sutton Coldfield tower near Birmingham. Because the radiation pattern was a Pattern B type they studied 20 other sites to see if they had the same results. All other sites individually and when grouped showed Pattern A types. However, no other site that Birmingham had both a high population close to the tower and a VHF high power emission. Hence all other sites logically produced Pattern A radial types, Figure 7.

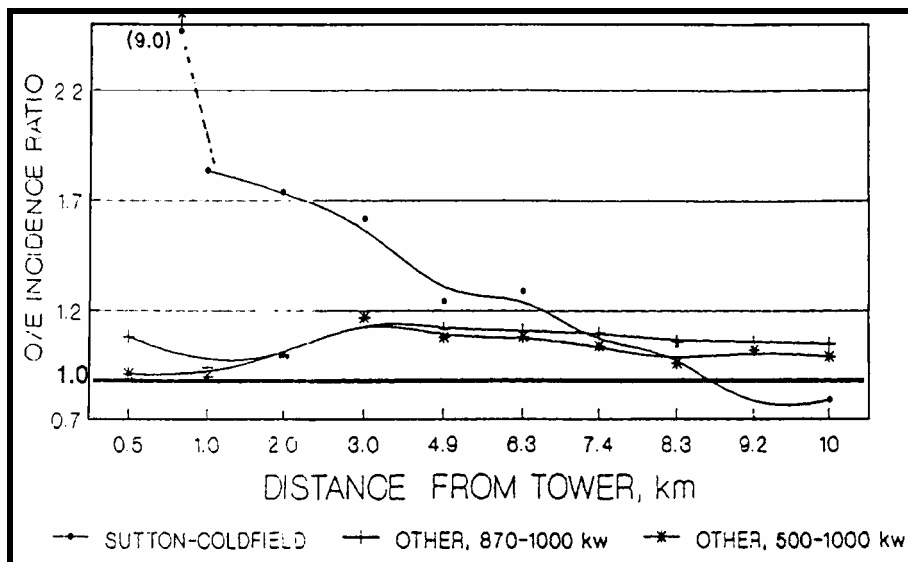


Figure 7: The radial patterns of adult cancers in Sutton Coldfield and 20 other regional TV/FM transmission sites in United Kingdom, from Goldsmith (1997) derived from Dolk et al. (1997a,b).

Cherry (2001) appropriately concludes that this makes the chance of confounding factors vanishing small and indicates a causal relation between a range of adult cancers and chronic exposure to very low mean-intensity RF radiation at less than 0.1% of present standards.

Antenna Radiation Patterns:

Around broadcast towers the ground level exposure patterns are a function of the power of the source signal and the antenna gain. The gain, expressed as a function of the Equivalent Isotropic Radiated Power (EIRP) is a function of the technology used to focus the signal. Antennae are complex elements that attempt to efficiently focus the main beam and minimize the side-lobes. The ability to do this to some extent is a function of the carrier frequency. Because of these side-lobes a complex antenna pattern is formed with undulating peaks in the 'near field' towers, which typically extends out to 5 to 6 km. Figures 8. VHF antenna patterns produce ground level Pattern A, with a high exposure peak with 1 km of the tower, Figure 8a.

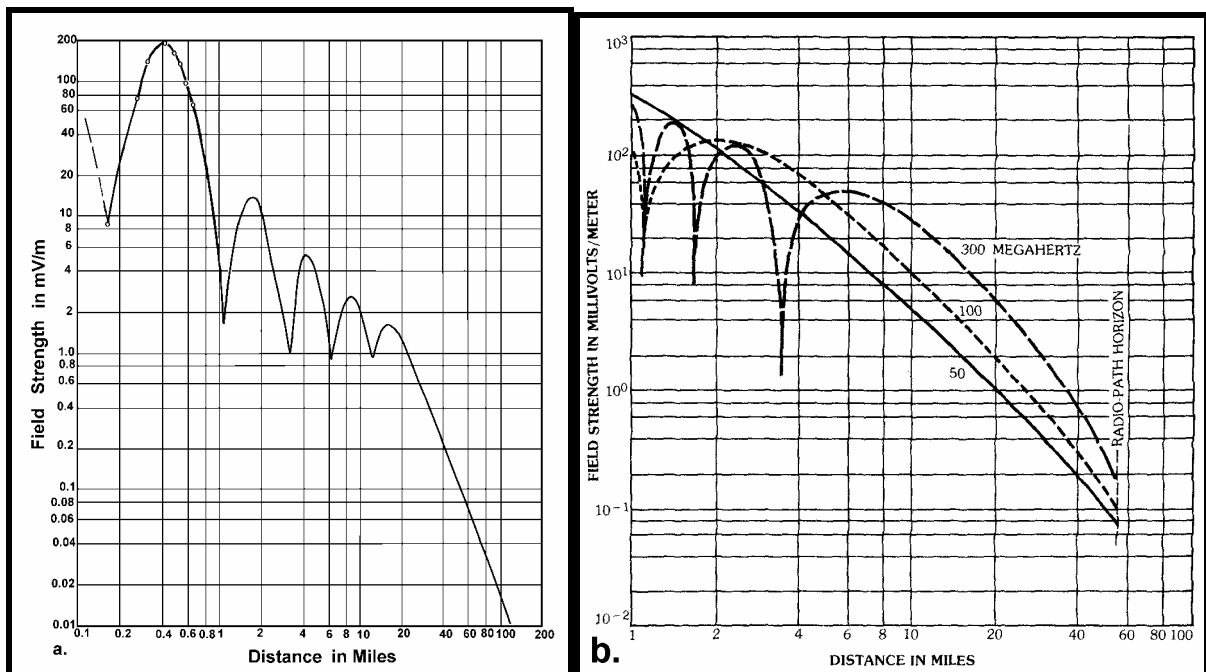


Figure 8: Ground level radiation pattern for (a) the 44 MHz (VHF) signal from the Empire State Building in New York City, Jones (1936) by merging his figures 6 and 8, and (b) a theoretical set of 1 kW antenna at a height 1000ft and a receiver at a height 30ft, Jordon (1985).

Figure 8a shows the measured radial pattern near ground level around the Empire State Building in the 1930's, formed by the VHF stations installed on its tower. Figure 8b, from 'Reference data for Engineers', Jordon (1985), shows the dependence on the distance of the peaks and troughs as a function of the carrier frequency. The higher frequencies, 300 MHz, have higher relative peaks further out and lower relative peaks closer in than the 50 and 100 MHz signals. Note that the closest part of Figure 8b, is 1 mile (1.6 km) from the tower. Figure 8a shows for a 44 MHz signal, the highest peak at 0.4 miles, 640m.

Once the horizontal and vertical antenna patterns are known, the ground level exposure is a function of the gain for the particular elevation angle involved and the distance from the antenna, since the inverse square law operates along the ray of the beam. There are also signal strength variations caused by positive and negative phase reinforcement of the direct beam and the reflected beam at any point.

Vertical Antenna Patterns:

The vertical antenna pattern is a function of the antenna type and the carrier frequency. Figure 9 shows the vertical antenna pattern of an 8-dipole array for a 98 MHz FM station.

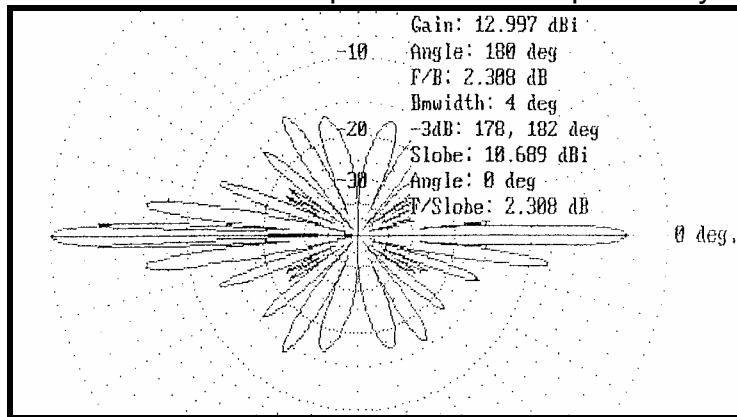


Figure 9: A typical vertical antenna pattern for a 4-element dipole array at about 98 MHz.(VHF), Units in dB.

The radial scale in Figure 9 is in dB that vary logarithmically with intensity. There is a very large difference between the intensity in the main horizontal beam (0 deg), the first minimum and the first side-lobe. These three points are -2.3, -28 and -8.1 dB respectively. These correspond to gains of 0.588, 0.00016 and 0.155, or relative gains of 1.0, 0.00027 and 0.2 respectively. The side-lobes have elevation angles of 8, 15, 40, 57 and 72 degrees. For an antenna at 500 m above ground level, the ground level side-lobe peaks occur at 160, 390, 600, 1870 and 3560 m from the base of the tower, with significant troughs of low exposure between them.

The amplitudes of the peaks and troughs are very large because of the logarithmic nature of the dB units. It is common to tilt the antenna pattern slightly downwards so that the main beam is directed towards major population centres in the listening and viewing area rather than towards the far horizon. The elevation angle of the antenna is usually slightly tilted downwards to point the main beam at the more remote listening or viewing audience. Figure 10 shows the relative antenna pattern for a UHF antenna with a down tilt of 0.5° . In contrast to the VHF signals in Figure 8 the UHF antenna patterns are more focussed towards the horizon, Figure 10.

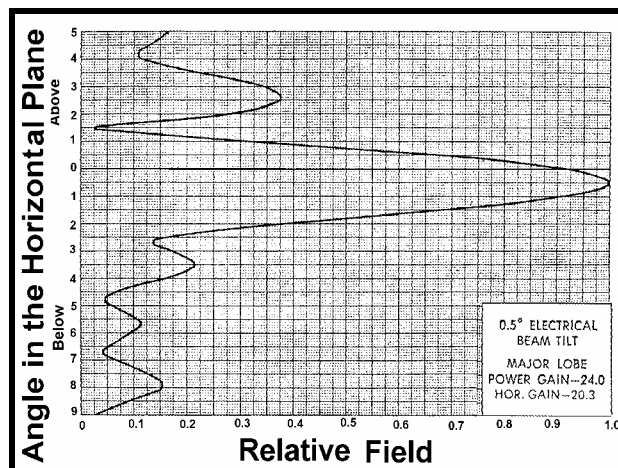


Figure 10: A typical Relative Field for a UHF RF/MW broadcast antenna from Hammett and Edison (1997).

The signal intensity is a function of the square of the Relative Field. The Relative Field peaks in Figure 10 are at 0.5, 3.5, 5.7, 7.9, 10.1 and 12.3°. With the assumption of the mean height of the antennae at 460m, these peaks correspond to ground level positions at 52.7, 7.5, 4.6, 3.3, 2.6 and 2.1km from the tower. The actual exposure intensity is a function of the square of the Relative Field and the inverse square of the distance along a beam. This results in the ground level peaks being closer to the Tower, especially for the most remote peaks. These adjustments are taken into account by the radial UHF pattern in Figure 10. This shows the main beam peaks at 12.5km and the major side-lobe peaks at 6, 4.5, 3.2, 2.2 and 1.1km.

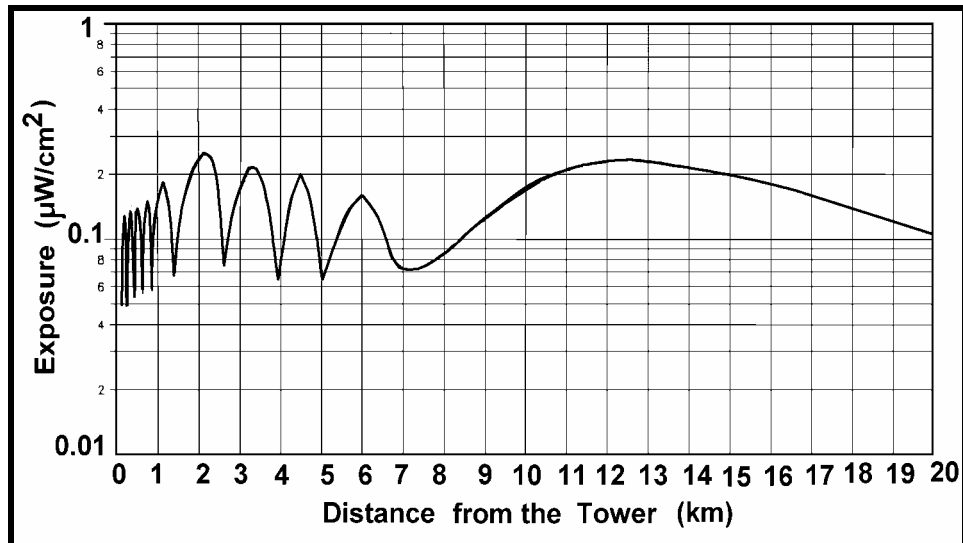


Figure 11: Ground level exposure for a typical UHF TV broadcast signal, from an antenna pattern (14), for an 18 MW EIRP transmitter at 460m AGL, for a flat horizontal surface.

Figure 11 shows the UHF antenna giving a Pattern A radial exposure pattern.

Horizontal antenna patterns:

Antennae are not only capable of focussing RF radiant power into vertical beams but can also focus the beams in the horizontal plain to send most of the broadcast signal towards most of the listeners and viewers. Two examples are given in Figures 12 and 13. The first is for the FM radio signal shown in Figure 9. The second is for the UHF antenna, the vertical pattern of which is in Figure 10.

Figure 12 shows that the signals from this antenna are horizontally focussed towards the city of Christchurch from the tower which is located to the northeast of the city.

The Sutro tower is in the western portion of the San Francisco Peninsula, with a small number of seaside suburbs behind it, most of the City of San Francisco, plus Oakland and Berkeley to the east.

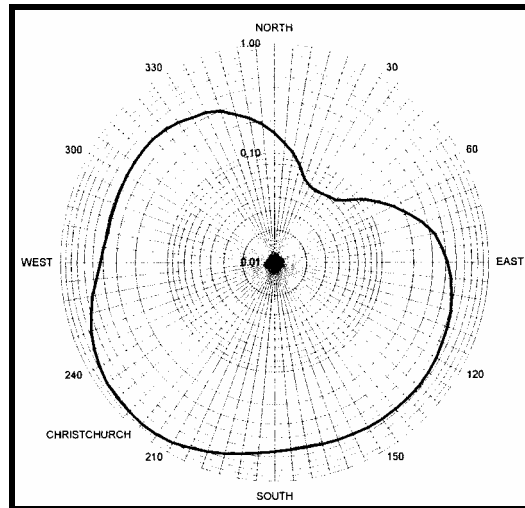


Figure 12: Horizontal antenna pattern for an 8-element dipole array for a 98 MHz FM transmission pointed towards the targeted high population city of Christchurch.

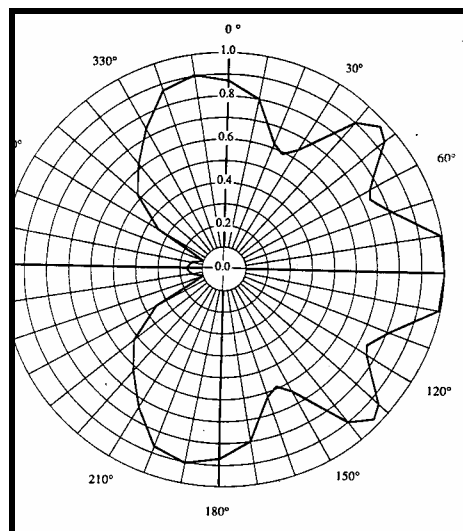


Figure 13: Horizontal antenna radiation patterns showing the relative field strength for, (a) UHF Digital TV (linear scale) from the Sutro Tower, Hammett and Edison (1997).

Both Figures 12 and 13 show how broadcast engineers can and do focus the signal to send it to the main receiver populations.

Adult and Childhood cancer in the Vicinity of Broadcast Towers:

There are now eleven published studies of residential adult and childhood cancer in the vicinity of radars, radio and TV transmission towers, from Kansas, U.S. Counties, Hawaii, Australia, United Kingdom, San Francisco and Rome. These are very important because they show that cancer is caused by very low-level RF/MW exposure well below the Western heating tissue based protection standards. The exposure levels definitely don't involve tissue heating and need another biological mechanism, for example genotoxic agency.

Wichita, Kansas:

The Lester and Moore (1982a) study shows a dose-response increase in all cancer with residential radar exposure, Figure 2.

U.S. Counties:

They then tested the hypothesis that radar caused cancer by investigating the cancer rates of counties with Air Force Bases (AFBs), because of their radar systems. Lester and Moore (1982b) found a significant elevation of cancer in males and females and all cancer in counties with AFBs. Polson and Merritt (1985) pointed out that a better method was to compare actual cities with proximity to AFBs. Lester (1985) adjusted their data accordingly and concluded that it strengthened their results.

Hawaii:

Maskarinec, Cooper and Swygert (1994) investigated a cluster of 12 children diagnosed with acute leukaemia in a section of the Waianae Coast, Hawaii. A matched case-control study was carried out to adjust the effects of parental occupational exposure, X-ray exposure, smoking, and previous sickness. When adjusted the Odds Ratios for childhood leukaemia for living within 2.6 miles of broadcast towers was OR = 2.0 (0.6-8.3) for the last residence before diagnosis, OR = 2.2 (0.3-15) residence at birth and OR = 1.8 (0.5-6.3) for maximum residence time.

Australia, North Sydney:

The concern for the potential effects of cell site base stations in Australia, Hocking et al. (1996) studied the cancer rates in the vicinity of three Radio and TV broadcast towers in North Sydney. They found that for the three municipalities adjacent to the towers the adult and childhood leukaemia incidence and mortality rate was significantly elevated. For the total population the incidence Rate Ratio was RR = 1.24 (1.09-1.40) and for children RR = 1.58 (1.07-2.34). For lymphatic leukaemia mortality in children RR = 2.74 (1.42-5.27), Tables 7 and 8.

Table 7: Rate Ratios (RR) and 95% confidence intervals (CI) for cancer incidence and mortality in the population of the inner area compared to the outer area, adjusted for age, sex and calendar period.

Cancer Type	RR (95% CI)	Cases
Incidence		
Brain Tumour	0.89 (0.71-1.11)	740
Total Leukaemia	1.24 (1.09-1.40)	1206
Lymphatic Leukaemia	1.32 (1.09-1.59)	536
Myeloid Leukaemia	1.09 (0.91-1.32)	563
Other Leukaemia	1.67 (1.12-2.49)	107
Mortality		
Brain Tumour	0.82 (0.63-1.07)	606
Total Leukaemia	1.17 (0.96-1.43)	847
Lymphatic Leukaemia	1.39 (1.00-1.92)	267
Myeloid Leukaemia	1.01 (0.82-1.24)	493
Other Leukaemia	1.57 (1.01-2.46)	87

Table 8: Rate Ratios (RR) and 95% confidence intervals (CI) for cancer incidence and mortality in childhood (0-14 years) in the population of the inner area compared to the outer area, adjusted for age, sex and calendar period.

Cancer Type	RR (95% CI)	Cases
Incidence		
Brain Tumour	1.01 (0.59-2.06)	64
Total Leukaemia	1.58 (1.07-2.34)	134
Lymphatic Leukaemia	1.55 (1.00-2.41)	107
Myeloid Leukaemia	1.73 (0.62-14.81)	9
Other Leukaemia	1.65 (0.33-8.19)	8
Mortality		
Brain Tumour	0.73 (0.26-2.10)	30
Total Leukaemia	2.32 (1.35-4.01)	59
Lymphatic Leukaemia	2.74 (1.42-5.27)	39
Myeloid Leukaemia	1.77 (0.47-6.69)	11
Other Leukaemia	1.45 (0.30-6.99)	9

The authors search diligently for confounding factors, including social economic factors, air pollution (benzene), ionizing radiation, migration, hospitals, high voltage power lines and local industries. None affected the relationships found. They investigated the possibility of clustering and found that no significant heterogeneity was found ($p=0.10$ for incidence and $p=0.13$ for mortality).

A follow-up study, McKenzie, Yin and Morrell (1998), compared the municipality rates with a calculated estimate of the mean centroid RF exposure level. This revealed a weak dose-response based on these assumptions, Figure 15.

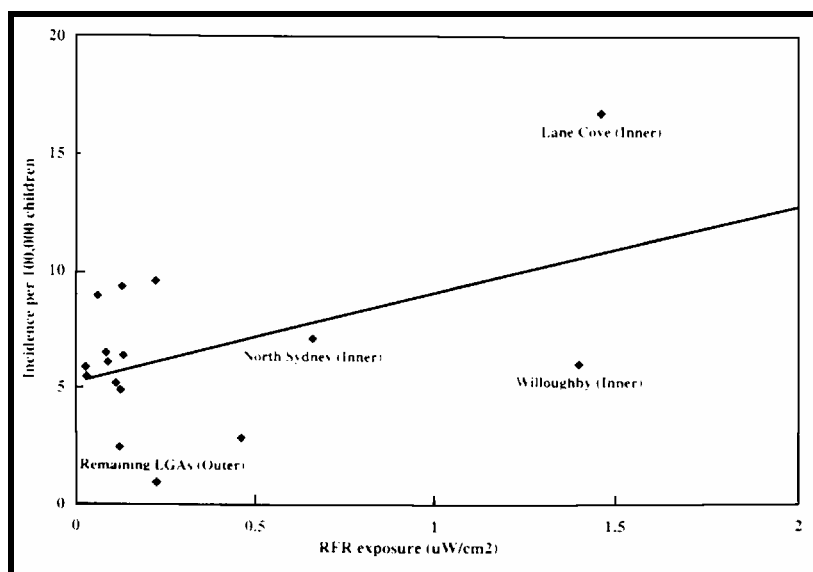


Figure 15: Incidence of total childhood leukaemia 1972-1990, versus theoretically calculated RF exposure from TV broadcast towers in North Sydney, for local government areas.

If the horizontal antenna patterns are taken into account, with the signals being directed towards the largest populations to the SE of the sites, it increases the exposure to Lane Cove, the area with the highest cancer rate, and decreases the mean exposure for Willoughby to less than North Sydney and strengthens the dose-response fit.

United Kingdom, Birmingham:

Dolk et al. (1997a,b) are discussed above showing exposure patterns matching adult leukaemia rates with exposure patterns, i.e. a dose-response relationship, Figure 7. Dolk (1997a) also investigated other cancers and found that outside 1.5 km all cancers except leukaemia followed a Type A Pattern, including All Cancer, Skin Melanoma and Bladder Cancer. Within 2 km of the Sutton Coldfield tower there were elevated rates of Adult Malignant and Benign Brain Cancer, Skin Melanoma, Male and Female Breast Cancer, Colorectal, Prostate and Bladder Cancer. For the high-powered tower groups the adult leukaemia rate followed the expected Pattern A and was still slightly elevated at 10 km.

A follow-up Sutton Coldfield study, Cooper, Hemmings and Saunders (2001) used the leukaemia rates for the period 1987-1994. They show only the 0-2km and 0-10km cancer rates. The results for All Leukaemia are summarised in Table 9.

Table 9: Summary of all leukaemia results from the follow-up Sutton Coldfield radio and TV tower, Cooper, Hemmings and Saunders (2001).

Exposed Group	Cases	0-2km		0-10km		
		O/E	95%CI	Cases	O/E	95%CI
Adult Females	8	1.23	0.53-2.42	159	1.26	1.07-1.46
Adult Males	12	1.40	0.72-2.44	174	1.09	0.93-1.25
All Adults	20	1.32	0.59-2.92	114	1.19	0.97-1.40
Child Females	0			11	1.13	0.57-2.03
Child Males	1	2.34	0.07-13.03	15	1.04	0.59-1.73
All Children	1	1.13	0.03-6.27	26	1.08	0.71-1.59

Despite the relatively small numbers the All Adult and All Children data shows a dose-response with higher rates in the 0-2km Group than the 0-10km Group.

San Francisco:

The Sutro Tower has high-powered VHF and UHF transmitters and a dense population living near the tower. Hence from the principles set out above it is predicted that there should be Pattern B cancer patterns, especially for leukaemia and brain cancer. This was confirmed using 0.5 km radial rings, Figures 16 and 17.

Figures 16 and 17 show the 0.5 km Risk Ratios for All cancer and Brain Cancer, respectively. They show high rates inside 1 km as shown in Dolk et al. for Birmingham.

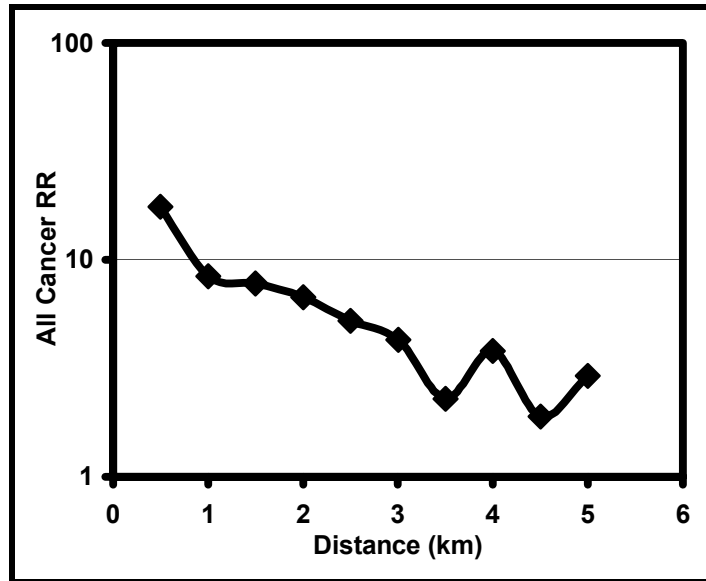


Figure 16: The 0.5 km band All Cancer Risk Ratio distance variation, using the >6km group as the control group.

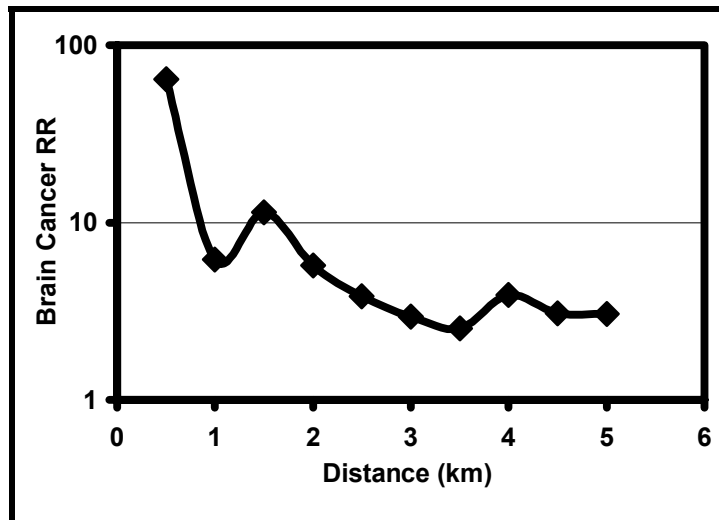


Figure 17: The 0.5 km band Brain Cancer Risk Ratio distance variation, using the >6km group as the control group.

Figures 16 and 17 show very similar type Pattern B. All Cancer data only has brain cancer in the <0.5km ring, hence it is exceptionally low when it included in the All Cancer data, Figure 16. Therefore the first point in the All Cancer data is reasonably treated as an outlier point.

Figure 18 sets out the 10 measured exposures in the 2 km radius, pointing to three peak and two minimum levels. The estimated exposure levels >2km are based on the UHF antenna pattern, Figure 11. The mixed VHF/UHF set of powerful antenna signals produce a Pattern B shape. The patterns are presented in logarithmic exposures and RR levels. The radial cancer rates are very similar to the exposure patterns. Both are Pattern B shapes. Hence the cancer radial patterns are close to the RF/MW probable radial pattern.

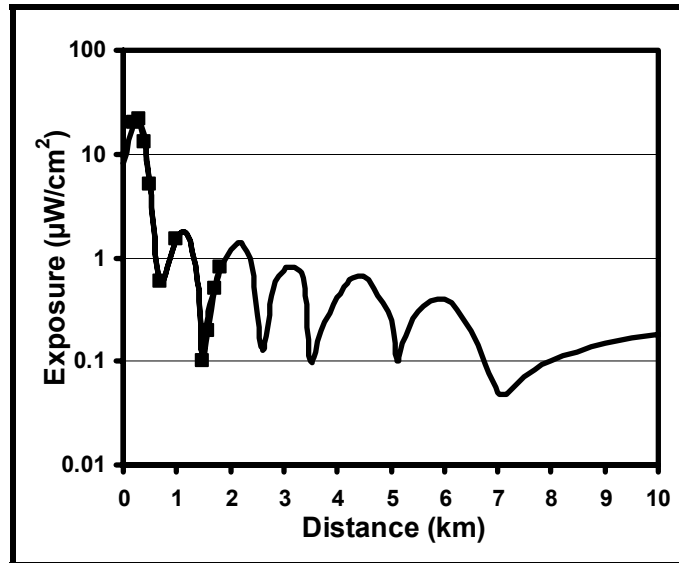


Figure 18: The measured and calculated radial exposure pattern from VHF and UHF antenna vertical patterns, Hammett and Edison (1997).

An exposure dose-response relationship is necessary for determining safety exposure standards, (18). Determining a 0.5km ring mean exposure pattern, noting the logarithmic scale and the observation that within 500m of the tower the houses are generally in the range 300-500m, with more homes at the larger radius, giving a mean exposure estimate of $8\mu\text{W}/\text{cm}^2$. Mean exposures for each 0.5km ring was taken from figure 11 and the 0.5km ring cancer rate was plotted in a dose-response graph. Figure 19 shows the All Cancer rate. The 0.1-0.5km ring only had the brain cancer in a small population group so it forms an outlier. The trend line was fitted to ignore the outlier. The Brain Cancer (Figure 20) and Leukaemia/ Lymphoma (Figure 21) trends do not show outliers but for the Leukaemia/ Lymphoma there are no cases for the $8\mu\text{W}/\text{cm}^2$ (0.1-0.5km) ring.

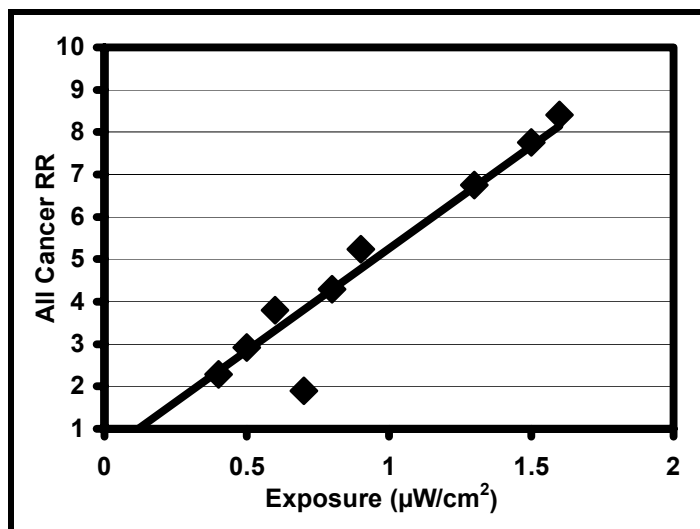


Figure 19: All Childhood Cancer as a function of measured and estimated RF/MW exposure in 0.5 km radial rings, ignoring the $9\mu\text{W}/\text{cm}^2$ outlier that only contained Brain Cancer. Trend $p < 0.0001$.

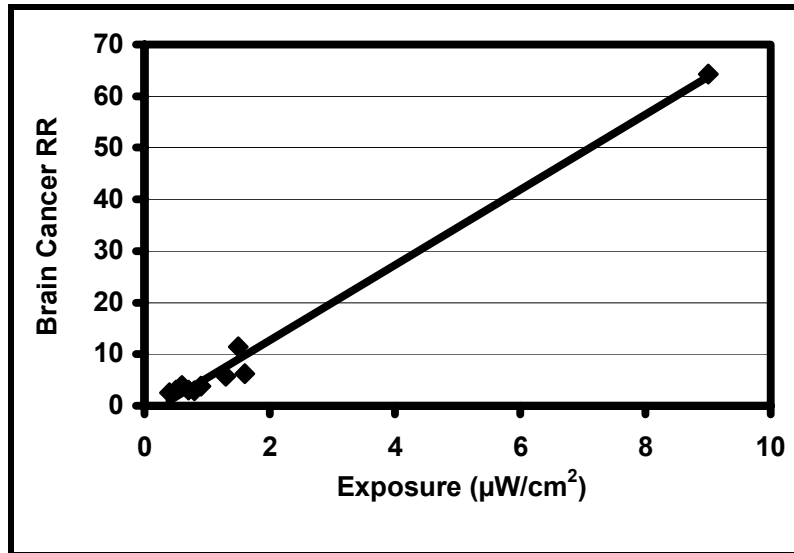


Figure 20: Childhood Brain Cancer as a function of measured and estimated RF/MW exposure in 0.5 km radial rings. Trend $p < 0.00001$.

Hence the Sutro Tower significantly elevates childhood cancer in a dose-response manner in San Francisco. Actual mean exposures are conservatively estimated to be 20 times less than the direct exposure, Cherry (2002).

Vatican Radio Towers Cancer Trends:

The Vatican radio antenna farm to the west of Rome has many powerful antennas with a wide of frequencies covering the VHF and UHF ranges. Hence a Pattern B is expected.

From Michelozzi et al. (2002): "Mortality of leukaemia of the adult population and the incidence of childhood leukaemia in the area exposed to emissions of radiofrequency near the Vatican Radio Towers". N = number of cases. The results are in Figure 22 and 23.

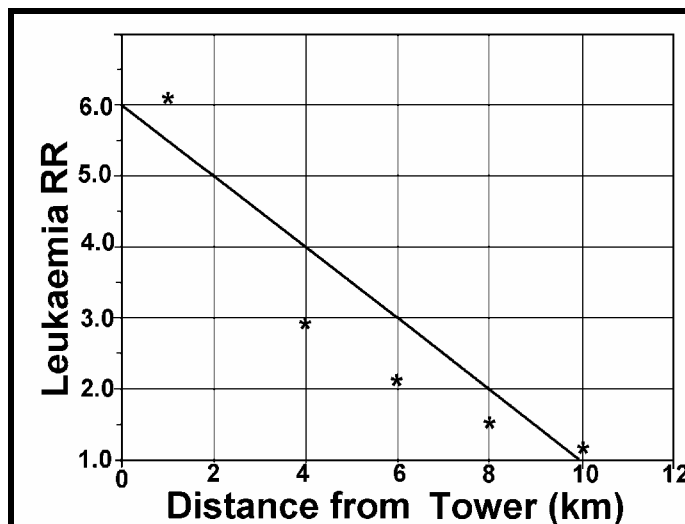


Figure 21: Radial cumulative childhood (<14yrs) leukaemia incidence in relation to distance from the Vatican Radio Towers, Rome, Michelozzi et al. (2002). Trend $p < 0.05$.

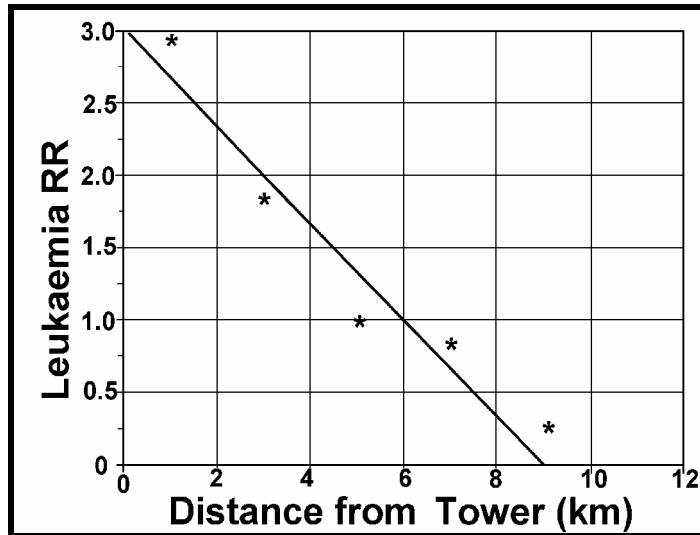


Figure 22: Adult leukaemia mortality in the vicinity of the Vatican Radio Towers, Rome, Michelozzi et al. (2002). Trend $p = 0.03$.

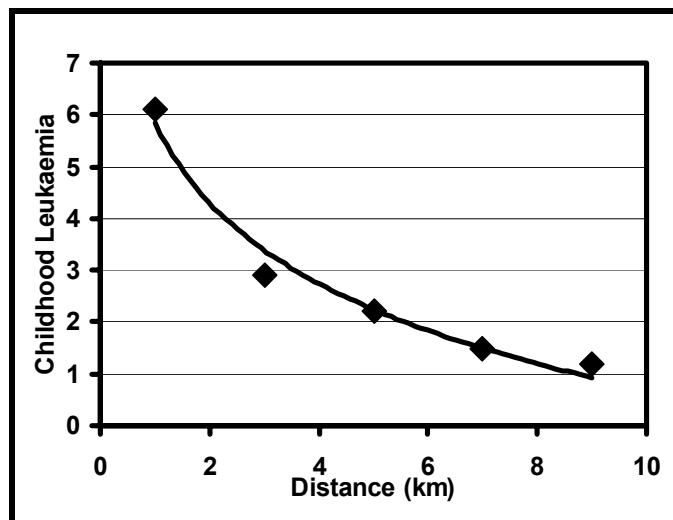


Figure 23: Cumulative childhood leukaemia near the Vatican Rome Radio Station with an exponential fitted trend line, $R^2=0.9756$, $p = 0.002$, Michelozzi et al. (2002).

The Vatican Radio study is the sixth epidemiological study to show dose-response elevation of cancer rates around broadcast transmission towers, when the classical assessment of a causal relationship, Hill (1965), can be based on only one. Six is far stronger and consistent evidence. It shows without doubt a causal relationship between a wide range of cancers and extremely low mean exposures to RF/MW exposure in the vicinity of radio and TV broadcast stations.

This is not surprising if you consider Table 10, a summary of the evidence of genotoxicity across the RF/MW spectrum. A genotoxic substance is a causal cancer agent, with no safe threshold. Therefore it is plausible and proven and people who with the vicinity of radio and TV towers show a dose-response increase in cancer, with the cancer rates getting higher the closer to the tower you live.

Table 10: Published evidence of genotoxic effects from RF/MW exposure as function of carrier frequency.

Frequency MHz	Modulation or pulsed	SAR W/kg	Genotoxic Activity	Association and Significance	Reference
10-200 kHz	VDT		MEL	p<0.05	Arnetz and Berg (1996)
27	80-180	iso	CA	Elev*	Heller and Teixeira-Pinto (1959)
27	CW	iso	CCP	*	Cleary, Cao and Liu (1996)
50	16Hz		EPOA		Romano-Spica et al. (2000)
154-162	Pulsed	iso	MN	p<0.01	Balode (1996)
88.5-950	Radio/TV	iso	Infertility in Mice+		Magras and Xenos (1997)
813.56	iDEN	0.0024	DNA	p<0.0001	Phillips et al. (1998)
836.55	TDMA	0.0026	DNA	p<0.0001	Phillips et al. (1998)
836.55	TDMA	0.0026	EPOA	p<0.05	Ivaschuk et al. (1997)
810-880	cellphones		MEL	p=0.04	Burch et al. (1997)
837	TDMA	5-10	MN	p<0.001	Tice et al. (2002)
954	GSM	1.56	CA/DNA		Verschaeve et al. (1994)
900	GSM		CA	Elev	Maes et al. (2001)
900	GSM	0.13-1.4	Cancer in Mice**		Repacholi et al. (1997)
960	GSM	0.00002	CCP*	DR	Kwee and Raskmark (1998)
1250-1350	Pulsed Radar		MN	p<0.001	Garaj-Vrhovac et al. (1999)
1200-1300	Pulsed Radar		CA,MN	p<0.001	Garaj-Vrhovac et al. (1990)
1250-1350	Pulsed Radar		CA**		Garaj-Vrhovac et al. (1993)
2450	CW		CA*	DR	Yao (1978)
2450	CW		Fetal Death**		Berman et al. (1980)
2450			Mutations*	Potent	Blevins et al. (1980)
2450	CW		CA	Elev*	Goud et al. (1982)
2450	CW		Abnormal Sperm		Goud et al. (1982)
2450	CW		CA	*	Yao (1982)
2450	CW		CA		Banerjee et al. (1983)
2450	CW		CA	DR	Manikowska-Czerska et al (1985)
2450	CW		Abnormal Sperm	DR	Beechey et al. (1986)
2450	CW	iso	MN	DR*	Maes et al. (1993)
2450	CW		DNA change		Sarkar, Ali and Behari (1994)
2450	Pulsed	0.6,1.2	DNA	DR	Lai and Singh (1995)
2450	CW/Pulsed	1.2	DNA	p<0.001	Lai and Singh (1996)
2450	CW	iso	CCP	*	Cleary, Cao and Liu (1996)
2450	Pulsed	1.2	DNA	p<0.001	Lai and Singh (1997)
2450	CW	1.0	MN	p<0.025	Vijayalaxmi et al. (1997)
2450	Pulsed	0.15-0.4	Cancer in Mice*		Chou et al. (1992)
2450			Cancer in Mice+		Szmigielski et al. (1988)
2550	CW		DNA resonance**		Sagripanti and Swicord (1986)
2550		iso	GE	p=0.002	Saffer and Profenno (1992)
4000			DNA resonance**		Sagripanti and Swicord (1986)
6600			DNA resonance**		Sagripanti and Swicord (1986)
7700		iso	CA	DR	Garaj-Vrhovac et al. (1991)
7700		iso	CD	DR	Garaj-Vrhovac et al. (1991)
7700	CW		CA	DR*	Garaj-Vrhovac et al. (1990)
7700	CW		CA**,MN**	DR*	Garaj-Vrhovac et al. (1992)
8750			DNA resonance**		Sagripanti and Swicord (1986)

You will see from Table 10 that the studies cover the frequencies and modulations used for cellphone radiation and hence cellphone radiation is genotoxic and is a causal agent for cancer. As a genotoxic carcinogen it also causes cardiac, reproductive and neurological diseases and death. One of the factors in Table 10 is melatonin reduction (MEL). This was included because melatonin is the most potent naturally occurring antioxidant used in our bodies to protect us from oxygen free radicals that can damage DNA. Hence reduced melatonin leads to cancer. It is also a highly active neurological agent controlling sleep. To confirm the health effects of cell sites, sleep disturbance is an appropriate sensitive bio-indicator for all of the CCRN effects.

RF/MW and Cell sites with Sleep Disturbance:

Altpeter et al. (1995) and Abelin (1999) report a causal association between residential short-wave radio exposure and sleep disturbance in Switzerland, Figure 24 and 25 show the dose-response relationship. Causation was based on dose-response, experimentation showing a significant alteration of sleep down to 0.4nW/cm^2 , Figure 26, and measured melatonin reduction.

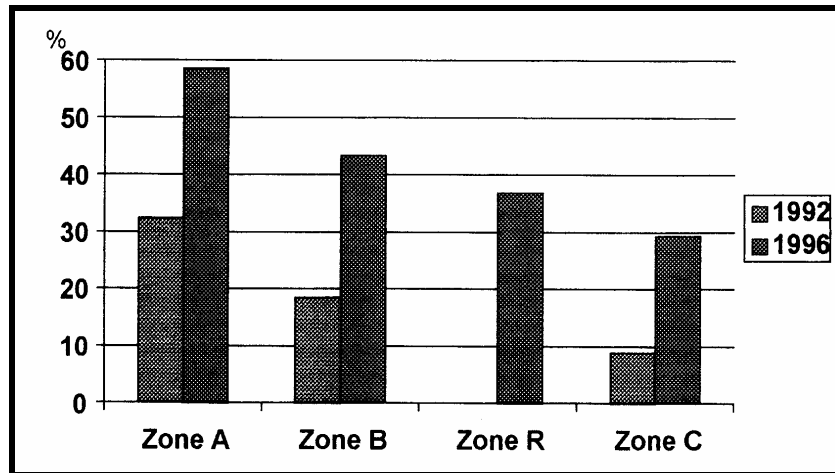


Figure 24: Adult Sleep Disturbance with RF exposure at Schwarzenburg, Switzerland, Abelin (1999).

Sleep disruption occurs in a dose-response manner with a threshold below 0.1nW/cm^2 , i.e. very close to zero, Figure 21.

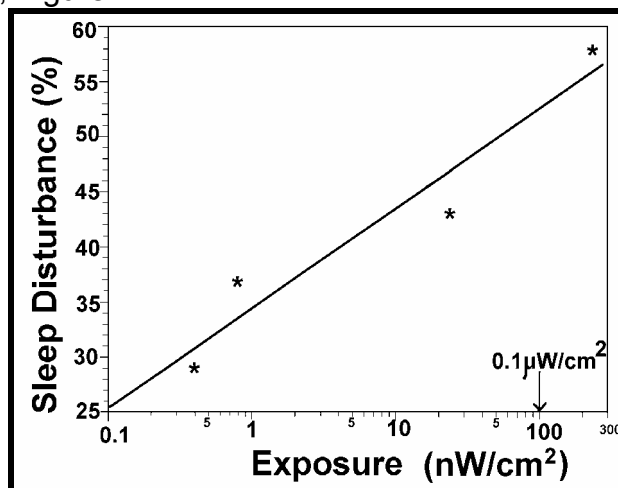


Figure 25: Dose-response relationship for Sleep Disturbance at Schwarzenburg with exposure in nW/cm^2 . Note: $1\text{nW/cm}^2 = 0.001\mu\text{W/cm}^2$

An experiment was carried out by secretly turning the tower off for three days. This showed significant melatonin increase in exposed cows and highly significant, $p < 0.001$, increase in sleep quality in all three groups, including Group C exposed to 0.4 nW/cm^2 .

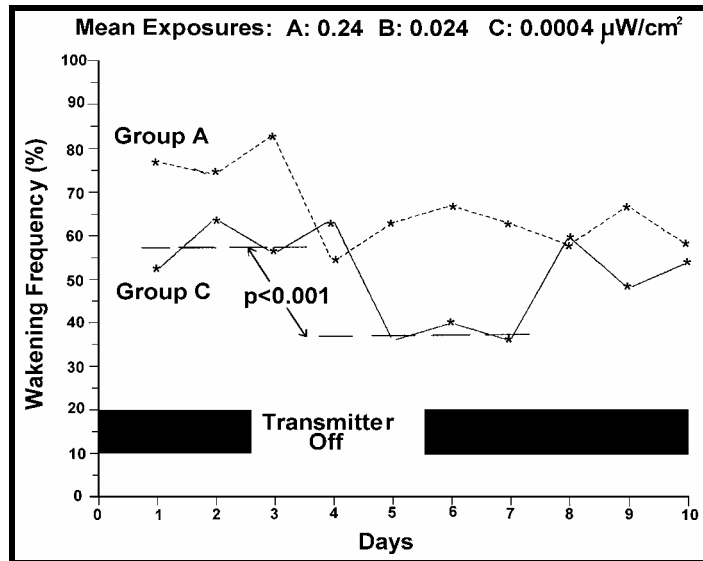


Figure 27: Sleep disturbance in people exposed to a short-wave radio stations which was turned off for three days, Altpeter et al. (1995), showing the highest exposed Group A, and lowest exposed Group C.

Both Groups show a delayed improvement in sleep of one to two days. The reduced wakening averaged over days 4 to 6 compared with days 1 to 3 are highly significantly reduced, $p < 0.001$. Thus the lowest exposed group, $0.0004 \mu\text{W/cm}^2$ also shows a significant effect of the RF exposure on sleep disturbance.

Thus turning the tower off revealed significant rises in bovine melatonin and human sleep quality. Human melatonin increased significantly when the tower was turned off permanently.

Groups B, R and C are all exposed to a mean RF signal of less than $0.1 \mu\text{W/cm}^2$ and they experienced highly significant sleep disturbance and reduced melatonin. Since sleep disturbance, Mann and Roschkle (1995), and melatonin reduction, has been observed with cell phone usage, Burch et al. (1997), then cellphone radiation will have the same effects. Hence these adverse health effects also result from exposure to cell phones and to radiation from cell sites. Santini and Santini (2002) have confirmed this by surveying symptoms around cell sites in France. A dose-response in sleep disturbance was a core result. The sleep disturbance, Figure 26, and other neurological health effects follow the typical cell site antenna radial pattern, Figure 27.

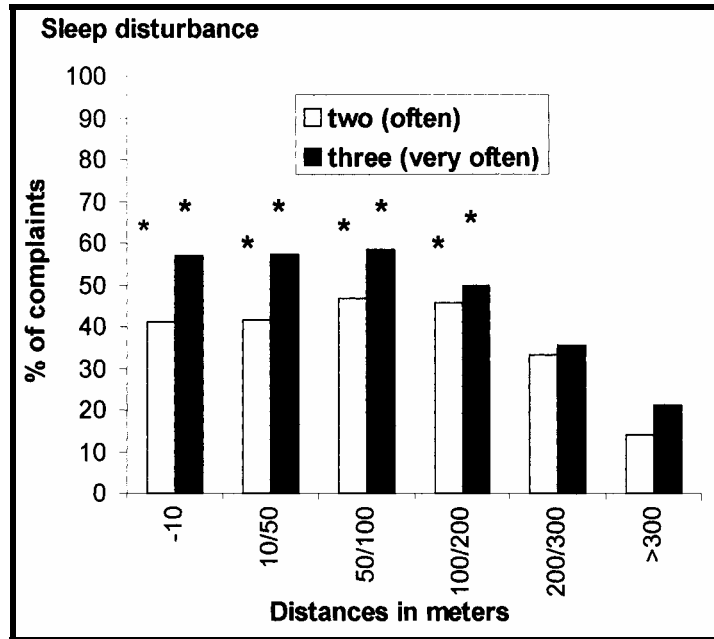


Figure 26: Percentages of complaints for sleep disturbance (responses 2 = Often and 3 = Very often) for men and women (530 subjects) in relation to distance from cellular phone base stations. * = $p < 0.05$ for comparisons with subject at a distance > 300 m or not exposed to base stations (control group), Santini and Santini (2001).

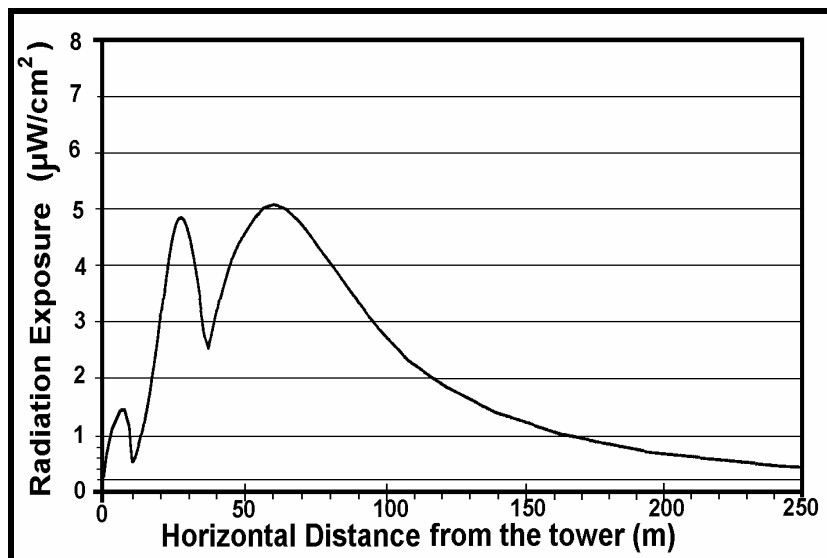


Figure 27: A typical cell site radial center main beam ground level (2m) radiation exposure level.

Conclusions:

There is robust evidence that EMR is genotoxic across the RF/MW spectrum, including radiation from radars, radio and TV stations, appliances, cellphones and cellphone base stations. To date over 50 studies have shown adverse biological or human health effects specifically from cell phone radiation.

The evidence presented here shows that EMR is a Ubiquitous Universal Genotoxic Carcinogen. That is, EMR exposes everyone and causes significant elevation of cancer

rates across many body organs, with no safe threshold. This is because of the combination of the DNA damaging biological mechanism of individual cells and the reduction of melatonin through a very sensitive brain interaction resonant process, reducing the cell damage repair rate and increasing the free radical damage rate.

These research results to date clearly show that cell phones and cell phone radiation are a strong risk factor for all of the adverse health effects identified for EMR because they share the same biological mechanisms. The greatest risk is to cell phone users because of the high exposure to their heads and the great sensitivity of brain tissue and brain processes. In the general population the combination of exposure to telecommunication signals, cell cordless phones, cellphone direct and passive exposure, cellphone bases station antennas saturating society, many disease and mortality rates are rising from these sources.

Because the biological mechanisms for cell phone radiation mimics that of EMR, and the dose-response relationships have a threshold of ZERO, and this includes genetic damage, there is extremely strong evidence to conclude that:

Cell sites radio/TV towers and radars are risk factors for:

- **Cancer across all body organs, especially brain tumour, breast cancer and leukaemia,**
- **Cardiac arrhythmia, heart attack and heart disease,**
- **Neurological effects, including sleep disturbance, learning difficulties, senility, depression and suicide.**
- **Miscarriage, congenital malformation, Sudden Infant Death syndrome, early childhood cancer, especially Acute Lymphoblastic Leukaemia (ALL).**

It is recommended to develop strategies, plans and laws to quickly and progressively reduce public exposures towards an initial mean chronic public exposure of

100 nW/cm².

In 5 years

10 nW/cm².

In 10 years.

References:

- Abelin, T., 1999: "Sleep disruption and melatonin reduction from exposure to a shortwave radio signal". Seminar at Canterbury Regional Council, New Zealand. August 1999.
- Adey, W.R., 1988: "Cell membranes: The electromagnetic environment and cancer promotion", *Neurochemical Research*, 13 (7): 671-677.
- Altpeter, E.S., Krebs, Th., Pflugler, D.H., von Kanel, J., Blattmann, R., et al., 1995: "Study of health effects of Shortwave Transmitter Station of Schwarzenburg, Berne, Switzerland". University of Berne, Institute for Social and Preventative Medicine, August 1995.
- Arnetz, B.B. and Berg, M., 1996: "Melatonin and Adrenocorticotrophic Hormone levels in video display unit workers during work and leisure. *J Occup Med* 38(11): 1108-1110.
- Aksoy, M., Erdem, S., DinCol, G., 1974: "Leukemia in shoe-workers exposed chronically to benzene". *Blood*. 44(6): 837-41.
- Aksoy, M., 1980: "Different types of malignancies due to occupational exposure to benzene: a review of recent observations in Turkey". *Environ Res. Oct*; 23(1): 181-90.
- Balode, Z., 1996: "Assessment of radio-frequency electromagnetic radiation by the micronucleus test in Bovine peripheral erythrocytes". *The Science of the Total Environment*, 180: 81-86.
- Banerjee R, Goldfeder A, Mitra J., 1983: "Combined effects of misonidazole, microwave hyperthermia, and X-rays on the *in vivo* sister chromatid exchanges and chromosomal abnormalities of the dbrB tumor and on the survival of its host". *Cancer Res* 43(5): 2210-2215.
- Baranski, S. and Czerski, P., 1976: "Biological effects of microwaves". Publ. Dowden, Hutchison and Ross, Inc. Stroudsburg, Pennsylvania.
- Berman, E., Carter, H.B. and House, D., 1980: "Tests of mutagenesis and reproduction in male rats exposed to 2,450 MHz (CW) microwaves". *Bioelectromagnetics* 1: 65-76.
- Beechey CV, Brooker D, Kowalczyk CI, Saunders RD Searle AG., 1986: "Cytogenetic effects of microwave irradiation on male germ cells of the mouse". *J Radiat Biol Relat Stud Phys Chem Med* 50(5): 909-918.
- Blevins, R.D., Crenshaw, R.C., Hougland, A.E. and Clarke, C.E., 1980: "The effects of microwave radiation and heat on specific mutants of *Salmonella typhimurim* LT2". *Radiation Research* 82: 511-517.
- Burch, J.B., Reif, J.S., Pittrat, C.A., Keefe, T.J. and Yost, M.G., 1997: "Cellular telephone use and excretion of a urinary melatonin metabolite". In: Annual review of Research in Biological Effects of electric and magnetic fields from the generation, delivery and use of electricity, San Diego, CA, Nov. 9-13, P-52.
- CCSFDPH, 1988: "Report on Cancer Incidence in Fan Francisco". City and County San Francisco Department of Public Health, 101 Grove Street, San Francisco, California.
- Cherry, N, 2001: Re: "Cancer incidence near radio and television transmitters in Great Britain, II All high power transmitters, Dolk et al. 1997 a,b in *American J. of Epidemiology*, 145(1):1-9 and 10-17. Comment in *American J of Epidemiology* 153(2): 204-205.

- Cherry, N.J., 2002: "Schumann Resonances, a plausible biophysical mechanism for the human health effects of Solar/Geomagnetic Activity". *Natural Hazards* (In Press).
- Chou, C-K., Guy, A.W., Kunz, L.L., Johnson, R.B., Crowley, J.J. and Krupp, J.H., 1992: "Long-term, low-level microwave irradiation of rats". *Bioelectromagnetics* 13: 469-496.
- Cleary, S.F., Cao, G. and Liu, L-M., 1996: "Effects of isothermal 2.45 GHz microwave radiation on the mammalian cell cycle: comparison with effects of 27 MHz radiofrequency radiation exposure". *Bioelectrochemistry and Bioenergetics* 39: 167-173.
- Cooper, D., Hemmings, K and Saunders P, 2001: Re: "Cancer incidence near radio and television transmitters in Great Britain, II All high power transmitters, Dolk et al. 1997 a,b in *American J. of Epidemiology*, 145(1):1-9 and 10-17. Comment in *American J of Epidemiology* 153(2): 202-204.
- De Longe, J.O., 1984: "Operant behaviour and colonic temperature of *Macaca mulatta* exposed to radiofrequency fields at and above resonant frequencies". *Bioelectromagnetics* 5: 233-246.
- Dolk, H., Shaddick, G., Walls, P., Grundy, C., Thakrar, B., Kleinschmidt, I. and Elliott, P., 1997a: "Cancer incidence near radio and television transmitters in Great Britain, I - Sutton-Colfield transmitter". *American J. of Epidemiology*, 145(1):1-9.
- Dolk, H., Elliott, P., Shaddick, G., Walls, P., Grundy, C., and Thakrar, B., 1997b: "Cancer incidence near radio and television transmitters in Great Britain, II All high power transmitters". *American J. of Epidemiology*, 145(1):10-17.
- Dutta, S.K., Subramoniam, A., Ghosh, B. and Parshad, R., 1984: "Microwave radiation-induced calcium ion efflux from human neuroblastoma cells in culture." *Bioelectromagnetics* 5(1): 71-78.
- Fishbeck, W.A., Townsend, J.C. and Swank, M.G., 1978: "Effects of chronic occupational exposure to measured concentrations of benzene". *J Occup Med.* 20(8): 539-42.
- Garaj-Vrhovac, V., Fucic, A, and Horvat, D., 1990: "Comparison of chromosome aberration and micronucleus induction in human lymphocytes after occupational exposure to vinyl chloride monomer and microwave radiation". *Periodicum Biologorum*, Vol 92, No.4, pp 411-416.
- Garaj-Vrhovac, V., Horvat, D. and Koren, Z., 1990: "The effect of microwave radiation on the cell genome". *Mutat Res* 243: 87-93.
- Garaj-Vrhovac, V., Horvat, D. and Koren, Z., 1991: "The relationship between colony-forming ability, chromosome aberrations and incidence of micronuclei in V79 Chinese Hamster cells exposed to microwave radiation". *Mutat Res* 263: 143-149.
- Garaj-Vrhovac, V. Fucic, A, and Horvat, D., 1992: The correlation between the frequency of micronuclei and specific aberrations in human lymphocytes exposed to microwave radiation in vitro". *Mutation Research* 261: 161-166.
- Garaj-Vrhovac, V., Fucic, A. and Pevalek-Kozlina, B. 1993: "The rate of elimination of chromosomal aberrations after accidental exposure to microwave radiation". *Bioelectrochemistry and Bioenergetics*, 30: 319-325.
- Garaj-Vrhovac, V. 1999: "Micronucleus assay and lymphocyte mitotic activity in risk assessment of occupational exposure to microwave radiation". *Chemosphere* 39(13): 2301-2312.

- Goldsmith, J.R., 1997a: "Epidemiologic evidence relevant to radar (microwave) effects". *Environmental Health Perspectives*, 105 (Suppl 6): 1579-1587.
- Goldsmith, J.R., 1997b: "TV Broadcast Towers and Cancer: The end of innocence for Radiofrequency exposures". *Am. J. Industrial Medicine* 32 : 689-692.
- Goswami, P.C., Albee, L.D., Parsian, A.J., Baty, J.D., Moros, E.G., Pickard, W.F., Roti Roti, J.L. and Hunt, C.R., 1999: "Proto-oncogene mRNA levels and activities of multiple transcription factors in C3H 10T 1/2 murine embryonic fibroblasts exposed to 835.62 and 847.74 MHz cellular telephone communication frequency radiation". *Radiat Res* 151(3): 300-309.
- Goud, S.N., Usha Rani, M.V., Reddy, P.P., Rao, M.S. and Saxena, V.K., 1982: "Genetic effects of microwave radiation in mice". *Mutation Research* 103: 39-42.
- Haider, T., Knasmueller, S., Kundi, M, and Haider, M., 1994: "Clastogenic effects of radiofrequency radiation on chromosomes of *Tradescantia*". *Mutation Research*, 324:65-68.
- Hammett and Edison Inc., 1997: "Engineering analysis of radio frequency exposure conditions with addition of digital TV channels". Prepared for Sutra Tower Inc., San Francisco, California, January 3, 1997.
- Hardell, L, Reizenstein, J, Johansson, B, Gertzen, H, Mild, KH, 1999: Angiosarcoma of the scalp and use of a cordless (portable) telephone. *Epidemiology* 10(6):785-786.
- Hardell, L, Nasman, A, Pahlson, A, Hallquist, A, Hansson Mild, K, 1999: Use of cellular telephones and the risk for brain tumours: A case-control study. *Int J Oncol* 15(1):113-116.
- Hardell, L, Nasman, A, Hallquist, A, 2000: "Case-control study of radiology work, medical X-ray investigations and use of cellular telephones as risk factors". *J of General Medicine*. www.medscape.com/Medscape/GeneralMedicine/journal/2000/v02.n03/
- Hardell, L., Hansson Mild, K., Pahlson, A. and Hallquist, A., 2001: "Ionizing Radiation, Cellular telephones and the Risk for Brain Tumors" *European Journal of Cancer Prevention*, 10, pp-523-529.
- Hardell, L., Hallquist, A., Hansson, Mild, K., Carlberg, M., Pahlson, A. and Lilja, A. 2002a: "Cellular and Cordless Telephones and the Risk for Brain Tumors", *European Journal of Cancer Prevention*. In press (June 2002).
- Hardell, L., Hansson Mild, K. and Carlberg, M., 2002b: "Use of Cellular Telephones and the Risk for Astrocytomas" unpublished manuscript, submitted to *International Journal of Radiation Biology*.
- Hayes, R.B., Morris Brown, L., Pottern, L.M., Gomez, M., Kardaun, J.W.P.F., Hoover, R.N., O'Connell, K.J., Sutsman, R.E. and Nasser, J., 1990: Occupational and Risk for Testicular Cancer: A Case Control Study. *International Journal of Epidemiology*, 19, No.4, pp 825-831.
- Hayes, R.B., Yin, S.N., Dosemeci, M., et al., 1996: "Mortality among Benzene-exposed workers in China". *Environ Health Perspect* 104 (Suppl 6): 1349-1352.
- Heller J.H. and Teixeira-Pinto A.A., 1959: "A new physical method of creating chromosome aberrations". *Nature* 183 (4665): 905-906.

- Hill, A. B., 1965: "The Environment and Disease: Association or Causation?" Proc. Royal Society of Medicine (U.K.). 295-300.
- Hocking, B., Gordon, I.R., Grain, H.L., and Hatfield, G.E., 1996: "Cancer incidence and mortality and proximity to TV towers". Medical Journal of Australia, Vol 165, 2/16 December, pp 601-605.
- Infante, P.F., Rinsky, R.A., Wagoner, J.K. and Young, R.J., 1977: "Leukaemia in benzene workers". Lancet. Jul 9; 2(8028): 76-78.
- International Agency for Cancer Research (IARC), 1982: "Benzene classification as a human carcinogen" IARC V29, 1982.
- Ivaschuk, O.I., Jones, R.A., Ishida-Jones, T., Haggren, Q., Adey, W.R. and Phillips, J.L., 1997: "Exposure of nerve growth factor-treated PC12 rat pheochromocytoma cells to a modulated radiofrequency field at 836.55 MHz: effects on c-jun and c-fos expression". Bioelectromagnetics 18(3): 223-229.
- Jacobson, C.B., 1969: Progress report on SCC 31732, (Cytogenic analysis of blood from the staff at the U.S. Embassy in Moscow), George Washington University, Reproductive Genetics Unit, Dept. of Obstetrics and Gynecology, February 4, 1969.
- Johnson, C.C. and Guy, A.W., 1972: "Non-ionizing electromagnetic wave effects in biological materials and systems". Proc IEEE 60(6): 692-718.
- Jones, L.F., 1933: "A study of the propagation of wavelengths between three and eight meters. Proc. of the Institute of Radio Engineers 21(3): 349-386.
- Jordan, E.C., (Ed), 1985: "Reference data for engineers: Radio, Electronics, Computer and Communications, 7th Edition". Publ. Howard W. Sams & CO., Indianapolis.
- Kwee, S, Raskmark, P, 1997: Radiofrequency electromagnetic fields and cell proliferation. Presented at the Second World Congress for Electricity and Magnetism in Biology and Medicine, Bologna, Italy, June.
- Kwee, S. and Raskmark, P., 1998: "Changes in cell proliferation due to environmental non-ionizing radiation 2. Microwave radiation". Bioelectrochem Bioenerg 44: 251-255.
- Lai, H. and Singh, NP., 1995: "Acute low-intensity microwave exposure increases DNA single-strand breaks in rat brain cells". Bioelectromagnetics 16: 207-210.
- Lai, H. and Singh, NP., 1996: "Single- and double-strand DNA breaks in rat brain cells after acute exposure to radiofrequency electromagnetic radiation". Int. J. Radiation Biology 69 (4): 513-521.
- Lai, H., and Singh, NP., 1997b: "Melatonin and Spin-Trap compound Block Radiofrequency Electromagnetic Radiation-induced DNA Strands Breaks in Rat Brain Cells." Bioelectromagnetics 18: 446-454.
- Lester, J.R., and Moore, D.F., 1982a: "Cancer incidence and electromagnetic radiation". Journal of Bioelectricity, 1(1): 59-76.
- Lester, J.R., and Moore, D.F., 1982b: "Cancer mortality and air force bases". Journal of Bioelectricity, 1(1): 77-82.

- Lester, J.R., 1985: "Reply to: Cancer mortality and air force bases, a reevaluation". *Journal of Bioelectricity*, 4(1): 129-131.
- Lilienfeld, A.M., Tonascia, J., and Tonascia S., Libauer, C.A., and Cauthen, G.M., 1978: "Foreign Service health status study - evaluation of health status of foreign service and other employees from selected eastern European posts". Final Report (Contract number 6025-619073) to the U.S. Dept of State, July 31, 1978.
- Maes, A., Verschaeve, L., Arroyo, A., De Wagter, C. and Vercruyssen, L., 1993: "In vitro effects of 2454 MHz waves on human peripheral blood lymphocytes". *Bioelectromagnetics* 14: 495-501.
- Maes, A., Collier, M., Slaets, D., and Verschaeve, L., 1996: "954 MHz Microwaves enhance the mutagenic properties of Mitomycin C". *Environmental and Molecular Mutagenesis*, 28: 26-30.
- Maes, A., Collier M., Van Gorp, U., Vandoninck, S. and Verschaeve, L., 1997: "Cytogenetic effects of 935.2-MHz (GSM) microwaves alone and in combination with mitomycin C". *Mutat Res* 393(1-2): 151-156.
- Maes, A. Collier, M. and Verschaeve, L., 2001: "Cytogenetic effects of 900 MHz (GSM) microwaves on human lymphocytes". *Bioelectromagnetics* 22: 91-96.
- Magras, I.N. and Xenos, T.D., 1997: "RF radiation-induced changes in the prenatal development of mice". *Bioelectromagnetics*, 18: 455-461.
- Manikowska, E., Luciani, J.M., Servantie, B., Czerski, P., Obrenovitch, J. and Stahl A., 1979: "Effects of 9.4 GHz microwave exposure on meiosis in mice". *Experientia* 35(3): 358-390.
- Manikowska-Czerska E, Czerski P Leach WM., 1985: "Effects of 2.45 GHz microwaves on meiotic chromosomes of male CBA/CAY mice". *J Hered* 76(1): 71-73.
- Mann, K, Roschke, J, 1996: Effects of pulsed high-frequency electromagnetic fields on human sleep. *Neuropsychobiology* 33(1):41-47.
- Maskarinec, G. Cooper, J. and Swygert, L., 1994: "Investigation of increased incidence in childhood leukemia near radio towers in Hawaii: Preliminary observations". *J. Environ Pathol Toxicol and Oncol* 13(1): 33-37.
- McKenzie, D.R., Yin, Y. and Morrell, S., 1998: "Childhood incidence of acute lymphoblastic leukaemia and exposure to broadcast radiation in Sydney - a second look". *Aust NZ J Pub Health* 22 (3): 360-367.
- Michelozzi, P., Ancona, C., Fusco, D., Forastiere, F. and Perucci, C.A., 1998: "Risk of leukaemia and residence near a radio transmitter in Italy". *ISEE/ISEA 1998 Conference, Boston Mass. Paper 354 P., Abstract in Epidemiology* 9(4):S111.
- Michelozzi, P., Capon, A., Kirchmayer, U., Forastiere, F., Biggeri, A., barca, A. and Perucci, C.A., 2002: "Adult and childhood Leukaemia near a high-powered radio station in Rome, Italy". *Am J of Epidemiology* 155: 1096-1103.
- Milham S., 1985: "Mortality in workers exposed to electromagnetic fields. *Environ Health Perspectives* 62:297-300.
- Milham, S., 1988: "Increased mortality in amateur radio operators due to lymphatic and hematopoietic malignancies". *Am. J. Epidemiology*, Vol 127, No.1, pp 50-54.

- Ott, M.G., Townsend, J.C., Fishbeck, W.A. and Langner, R.A., 1978: "Mortality among individuals occupationally exposed to benzene". *Arch Environ Health*. 33(1): 3-10.
- Phillips, J.L., Ivaschuk, O., Ishida-Jones, T., Jones, R.A., Campbell-Beachler, M. and Haggren, W., 1998: "DNA damage in molt-4 T-lymphoblastoid cells exposed to cellular telephone radiofrequency fields in vitro". *Bioelectrochem Bioenerg* 45: 103-110.
- Pollack, H., 1979: "Epidemiologic data on American personnel in the Moscow Embassy", *Bull. N.Y. Acad. Med*, 55(11): 1182-1186.
- Pollack, H., 1979a: "The microwave syndrome", *Bull. N.Y. Acad. Med*, 55(11): 1240-1243.
- Polson, P. and Merritt, J.H., 1985: "Cancer mortality and air force bases: a reevaluation". *Journal of Bioelectricity*, 4(1):121-127.
- Repacholi, MH, Basten, A, Gebiski, V, Noonan, D, Finnie, J, Harris, AW, 1997: Lymphomas in E mu-Pim1 transgenic mice exposed to pulsed 900 MHZ electromagnetic fields. *Radiat Res* 147(5): 631-640.
- Rinsky, R.A., Young, R.J. and Smith, A.B., 1981: "Leukemia in benzene workers". *Am J Ind Med*. 2(3): 217-45.
- Robinette, C.D., Silverman, C. and Jablon, S., 1980: "Effects upon health of occupational exposure to microwave radiation (radar)". *American Journal of Epidemiology*, 112(1):39-53, 1980.
- Romano-Spica, V., Mucci, N., Ursini, C.L., Ianni, A. and Bhat, N.K., 2000: "Ets1 oncogene induction by ELF-modulated 50 MHz radiofrequency electromagnetic field". *Bioelectromagnetics* 21: 8-18.
- Sagripanti, J. and Swicord, M.L., 1976: DNA structural changes caused by microwave radiation. *Int. J. of Rad. Bio.*, 50(1), pp 47-50, 1986.
- Saffer, J.D. and Profenno, L.A., 1992: "Microwave-specific heating affects gene expression". *Bioelectromagnetics* 13: 75-78.
- Santini R, Seigne M, Bonhomme-Faivre L, Bouffet S, Defrasne E, and Sage M., 2002: "Symptoms experienced by users of digital cellular phones: a study of the French engineering school. *Electromagnetic Biology and Medicine*. 21(1): 81-88.
- Sarkar, S., Sher, A. and Behari, J., 1994: "Effect of low power microwave on the mouse genome: A direct DNA analysis". *Mutation Research*, 320: 141-147.
- Schwan, H.P. and Foster, K.R., 1980: "RF-field interactions with biological systems: electrical properties and biophysical mechanisms". *Proc IEEE* 68(1): 104-113.
- Selvin, S., Schulman, J. and Merrill, D.W., 1992: "Distance and risk measures for the analysis of spatial data: a study of childhood cancers". *Soc. Sci. Med.*, 34(7):769-777.
- Stodolnik-Baranska, W., 1967: "Lymphoblastoid transformation of lymphocytes in vitro after microwaves irradiation". *Nature* 214, April 1: 102-103.
- Szmigielski, S., Szudzinski, A, Pietraszek, A., et al., 1982: "Accelerated development of spontaneous and benzopyrene induced skin cancer in mice exposed to 2450 MHz microwave radiation". *Bioelectromagnetics*, 3: 179-191.

- Szmigielski, S., Bielec, M., Lipski, S., and Sokolska, G., 1988: "Immunological and cancer-related aspects of exposure to low level microwave and radiofrequency fields". In Marino (Ed), 'Modern Bioelectricity', Marcel Bekker, N.Y., pp 861-925.
- Szmigielski, S., 1996: "Cancer morbidity in subjects occupationally exposed to high frequency (radiofrequency and microwave) electromagnetic radiation". *Sci Total Env* 180: 9-17.
- Theriault, G., Goldberg, M., Miller, A.B., Armstrong, B., Guenel, P., Deadman, J., Imbernon, E., To, T., Chevalier, A., Cyr, D., et. Al., 1994: "Cancer risks associated with occupational exposure to magnetic fields among electric utility workers in Ontario and Quebec, Canada and France: 1970-1989". *Am J Epidemiology* 139(6): 550-572.
- Tice R, Hook G, McRee DI., 1999: "Chromosome aberrations from exposure to cell phone radiation". *Microwave News*, Jul/Aug (1999) p7.
- Tice, R.R., Hook, G.G., Donner, M., McRee, D.I. and Guy, A.W., 2002: "Genotoxicity of Radiofrequency Signals. I. Investigation of DNA damage and Micronuclei induction in cultured human blood cells". *Bioelectromagnetics* 23: 113-126.
- Timchenko, O.I., and Ianchevskaia, N.V., 1995: "The cytogenetic action of electromagnetic fields in the short-wave range". *Psychopharmacology Series*, Jul-Aug;(7-8):37-9.
- Tonascia, J.A. and Tonascia, S., 1969: "Hematological Study: progress report on SCC 31732", George Washington University, Department of Obstetrics and Gynecology, February 4, 1969.
- Thorpe JJ. 1974Epidemiologic survey of leukemia in persons potentially exposed to benzene. *J Occup Med.* 16(6): 375-82.
- Townsend, J.C., Ott, M.G. and Fishbeck, W.A., 1978: "Health exam findings among individuals occupationally exposed to benzene". *J Occup Med.* 20(8): 543-8.
- UKRCEP, 1998: United Kingdom Royal Commission on Environmental Pollution, 21st Report: Setting Environmental Standards. United Kingdom Government.
- Velizarov, S, Raskmark, P, Kwee, S, 1999: The effects of radiofrequency fields on cell proliferation are non-thermal. *Bioelectrochem Bioenerg* 48(1):177-180.
- Verschaeve, L., Slaets, D., Van Gorp, U., Maes, A. and Vanderkom, J., 1994: "In vitro and in vivo genetic effects of microwaves from mobile phone frequencies in human and rat peripheral blood lymphocytes". *Proceedings of Cost 244 Meetings on Mobile Communication and Extremely Low Frequency field: Instrumentation and measurements in Bioelectromagnetics Research.* Ed. D, Simunic, pp 74-83.
- Vignati, M. and Giuliani, L., 1997: "Radiofrequency exposure near high-voltage lines". *Environmental Health Perspectives*, 105 (Suppl 6): 1569-1573.
- Vijayalaxmi BZ, Frei MR, Dusch SJ, Guel V Meltz ML, Jauchem JR. 1997: "Frequency of micronuclei in the peripheral blood and bone marrow of cancer-prone mice chronically exposed to 2450 MHz radiofrequency radiation". *Rad Res* 147: 495-500.
- Yao KT., 1978: "Microwave radiation-induced chromosomal aberrations in corneal epithelium of Chinese hamsters". *J Hered* 69(6): 409-412.

Yao KT., 1982: "Cytogenetic consequences of microwave irradiation on mammalian cells incubated in vitro". J Hered 73(2): 133-138.

Zaret, M.M., 1977: "Potential hazards of hertzian radiation and tumors. NY State J Med,146-147.